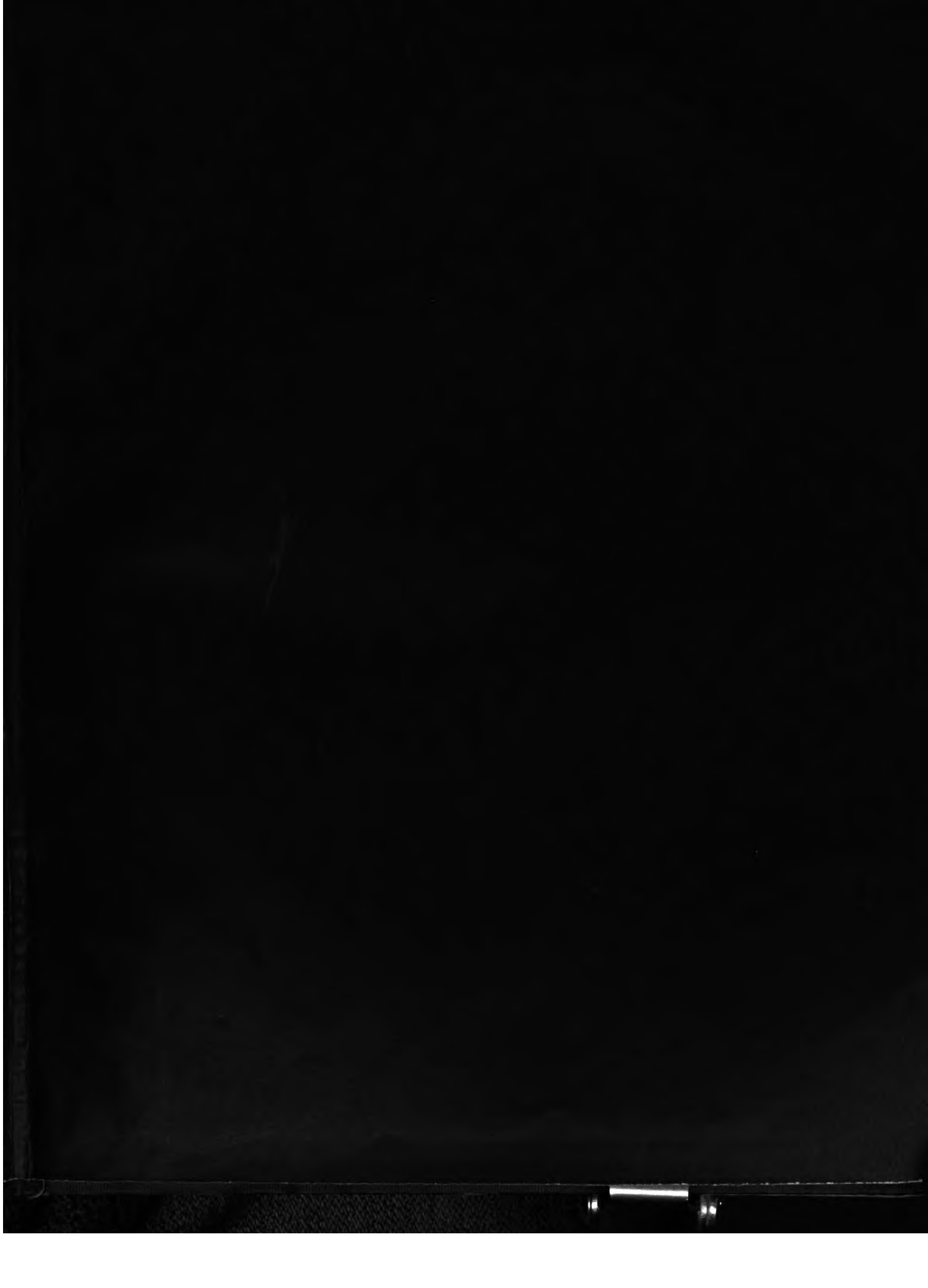

THE

Journal of Tropical Medicine

A MONTHLY JOURNAL DEVOTED TO MEDICAL, SURGICAL AND GYNÆCOLOGICAL WORK IN THE TROPICS

EDITED BY

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CAMP FEVERS.

By Dr. Filippo Rho.

Privat-docent of Medical Pathology (Univ. of Rome); Surgeon-Major of R. Italian Navy.

(*A continuation of Dr. Rho's article on Camp Fevers in the July Number*, 1900.)

Section II.

Observations in the United States and Cuba.

If we pass on to the reports of American doctors on the fevers treated among the militia during the Cuban war, we find that they tend to the same end as those made at Massowha, with the aggravating circumstance, however, of a complication, sometimes real and often hypothetical, caused by malaria.

In places where malaria really exists, the phantom of this infection often dims the judgment of the practitioner where he does not avail himself of all the modern diagnostic means. The desire to fix the differences between the typhoid and malarial infections has been an enticement to continued efforts for over a century; but only in these latter years has it been possible scientifically to diagnose both diseases by the aid of the microscopic and bacteriology. When these means cease to be the privilege of laboratories and clinics, and become the patrimony of every physician, only then will it be possible to correct a quantity of errors, that still bias the opinions and warp the statistics, with regard to the influence of climates and meteorological factors on the development of diseases.

This also recently happened to the physicians of the American militia, who were exceedingly preoccupied with this idea of malaria. In a brief *entre filet*, the *Boston Medical and Surgical Journal* (September 29, 1898), said: "There seem to be good reasons to believe that many, and perhaps most of the so-called cases of malaria among our troops in Florida, Cuba and Porto-Rico, are in reality attacks, not of malaria but of typhoid fever.

This laconic note is enlarged upon in an article by Dr. Vaughan, which appeared in the *American Journal of Medical Sciences* ("Some Remarks on Typhoid Fever among our Soldiers during the late War with Spain," July, 1899). The author and other bacteriologists had been commissioned by Surgeon-General Sternberg to hold an inspection in the various encampments, with a view to controlling the accuracy of the reports sent in at the War Office; these reports awoke a doubt about the doctors not having rightly diagnosed the fevers prevalent among the troops. The inspection in question based itself chiefly on tests of diagnosis by means of serum test, after Widal's method, and on a careful examination of the blood, with the object of discovering the malarial parasite.

Well, it was found that there also fevers were too lightly diagnosed malaria; and Vaughan thus concludes:—

"To summarise concerning the so-called protracted malarias reported by the regimental surgeons, we will say that, in our opinion, practically all of these were typhoid fever, and the following are our reasons for this opinion:—

"(1) The uneven distribution of the so-called malaria among regiments encamped side by side gives us cause to suspect that these were not malaria.

"(2) Some of the surgeons failed to record their cases as typhoid, and the assistant surgeons instead write in another report: 'The prevailing disease that

has caused the sickness we have had is typhoid fever.'

"(3) The results of several hundred blood examinations show that malaria was a very rare disease among the troops that remained in the United States.

"(4) Malaria, as it exists in this country, is easily controlled by moderate doses of quinine. All the so-called protracted malarias in our camps were treated with large doses of quinine and were not improved thereby. Consequently, we must conclude that the diagnosis given these cases was erroneous.

"(5) The mortality of the so-called protracted malarias corresponds with the mortality of typhoid fever, and furnishes most positive proof that these cases were not malarial.

"For the reasons already given, we have included all the protracted malarias among our list of the typhoids."

Typhoid fever was not only diagnosed malaria, but it was covered up by many other names—indigestion, dengue, intestinal disorder, continued fever, &c. In some regiments many cases were diagnosed simply common continued fever. In others, the surgeons seemed to regard enteric fever as distinct from typhoid fever. The belief of some of the medical men in "typho-malaria" is evident by their reports.

This aversion to calling typhoid fever by its right name seems to exist among medical officers in all armies. The German medical officers often call the disease "gastric fever," and this term appears now and then in our records when the surgeon happens to be a German. The French call it "manœuvre fever."

Dr. Vaughan says also that in his military experience typhoid fever is often apparently an intermittent disease. With regard to etiology he is quite confident, like ourselves at Massowha, that water infection had but a subordinate share in the spread of typhoid fever among the soldiers in camps, and thinks that the disease was transmitted from one person to another by the transference of some part of the fæces from the sick to the alimentary canal of the healthy.

It is evident that there, as well as at Massowha, there was great reluctance in admitting the prevalence of typhoid fever; and it is conceivable, too, that the aversion to formulate such a diagnosis was all the greater, as the fevers of short duration, which accompany the endemo-epidemics of typhoid fever, especially in hot countries, presented an enormous average.

On this head we have an interesting statement by Dr. John W. Ross, surgeon of the U.S. Navy, on the "Epidemic which prevailed at Key West" (*New York Medical Record*, December 24, 1898). It is of importance to notice what he incidentally observes, *i.e.*, that at Santiago, the seat of war, the predominant fevers were of the same nature. The main features this infection presented were:

(1) A long period of incubation, from twelve to fourteen days, calculated from the moment it could be presumed the contagion took place among the different individuals.

(2) A sudden invasion with or without shiverings, and slight premonitory symptoms in a few cases.

(3) A rapid rising of the temperature, up to 30·7°-40° C. at first, ending by lysis.

(4) Constant cephalalgia, especially in the forehead and occiput; at times pains of a rheumatic order.

(5) Constipation, generally moderate, rarely diarrhœa; a furry tongue, gastric disorders.

(6) A measle-like rash which appeared between the fifth and seventh days, in the 25 per cent. of cases, mostly on the chest, abdomen and extremities.

(7) A slight enlargement of the spleen, sleeplessness to a higher or lesser degree.

(8) Very little mortality; in fact, hardly any.

As is seen, the symptoms in these short-lived fevers and those observed in the pyrexia prevailing at Massowha are perfectly identical, and this identity is equally to be found in the curves of temperature noticed in these two distant lands.

As has been repeatedly stated, we are of opinion that such fevers are nothing but light abortive forms of typhoid fever. Our colleague of the United States, on the contrary, concludes that, excluding the diagnosis of yellow fever and dengue, it was an epidemic of exanthematic typhus. But such an opinion has no foundation, either clinically or epidemiologically speaking. The above-mentioned symptoms, such as the prevalence of gastric disorders and constipation, the scarcely-enlarged spleen, the absence of the characteristically hæmorrhagic spots (purple petechiæ), the course run by the fever, and decreasing gradually, the low average of mortality, are all so many special signs which cause us absolutely to reject the diagnosis of exanthematic typhus, whereas they remind us of a mild form of the typhoid. It is much to be regretted that at Key-West the Widal's reaction was not tried as it would have dispelled every doubt.

And it is precisely the large amount of slight cases of typhoid fever, as they appear in camps in tropical or sub-tropical countries, that has till now favoured so many contradictory and hypothetical notions on the nature of those epidemics, and caused even the most typical cases to be erroneously diagnosed. We have seen, however, that in the Spanish-American war the diagnosis was often rectified by special commissions of competent medical men, or else it was made on such patients as had suffered most and longest; and this happened with regard to the sick who were brought home and nursed in the hospitals at New York, Philadelphia, Boston, &c.

In fact, the Doctors Thomson, Cotton Park, Ewing, Wyncop, Hammack, &c., have made their reports on the most serious and protracted cases, which did not spontaneously exhaust themselves in a short time, nor get cured by the sole change of place, as did the same fevers prevailing at Key-West. In these cases, properly studied with the application of modern means, it was seen that they were most of them of a typhoid, malarial, or dysenteric order, and isolated; or more rarely of a mixed infection, typhoid and malarial, or else dysenteric and malarial.

All these authors reject the notion of the existence of a properly-called typho-malarial fever. According to them it is merely a question of simple co-existence or complication, which does not give birth to a new disease, any more than the co-existence of malaria and dysentery. The patients had in most cases been suffering from malaria, and evidenced the

symptoms of typhoid infection later. Generally the malaria parasite does not manifest itself during the active period of the typhoid fever, nor even during the first days of convalescence; it is afterwards that the malarial parasite makes its presence felt. According to Thomson, the malarial fever rarely breaks out while the typhoid is in course of development; but this sometimes does happen, and may deceive the doctor, who, imagining it to be an aggravation, is not aware that it is a complication in the disease.

Again, as is seen, these numerous observations on the so-called typho-malaria confirm that which Baccelli had asserted as far back as 1875, before the discovery of Laveran, denying any importance to the proposed denomination and to the opinions then put forth by Woodward. Thus are also corroborated the facts which Laveran ascertained by means of the microscope, at the very outset of his celebrated researches on the blood of patients affected with malaria or typhoid, and which led him to discover the parasite.

In a memoir of his on the "Combined Typhoid and Malarial Infection" (*The American Journal of the Medical Sciences*, January, 1899), Dr. Irving Philipps Lyon came to the same conclusion. In this paper he exposes a case he met with, and examines all the cases which were scientifically studied and ascertained to be of a mixed character, and of which that paper does not merely give us a collective account, as did other above-mentioned relations by different Americans on cases observed during the recent war, but a particularised history. Of such cases there were twenty-nine in all; in some of them the malarial symptoms were prevalent masking the enteric alterations; but in most the symptoms of the typhoid had the upper hand. In such cases mortality would be double what it is in simple typhoid; so that an aggravation in the disease is evident.

Also Philipps Lyon rejects the name and notion of malarial typhoid, and recognises that they have been the source of much uncertainty and many errors in diagnosis. As to the frequency of these combined infections, he holds them to be rare in temperate climates, but probably rightly thinks that they are rather frequent in hot ones[1]. Since the above-mentioned statements were made by Park and Thomson, the former met with mixed infection among the typhoid fevers originating in Cuba, in the proportion of 1·5 per cent., the latter in the proportion of about 8 per cent. But it will not do, nor is it possible, to generalise on that head; and it should be borne in mind that those observers only attended patients in serious and in perfectly well-ascertained cases after their return from the seat of war; whereas, at Key-West and Cuba, there were thousands of cases in which the sick promptly recovered, and the infection must have been, nearly always, simple typhoid.

[1] In Italy the co-existence of both malarial and typhoid infection is truly exceptional; cases of this kind are extremely rare, and none have been found during the two last years at the hospitals of Rome, where so many enteric and malarial fevers are treated, and where now the reaction of Widal is generally tested, and examinations of blood for malarial parasites is never omitted (Discussion on the Serum-diagnosis of Widal at the *Società Lancosiana degli Ospedali di Roma*, March 17 and April 21, 1900).

Section III.

Malaria and Typhoid Infection.

In speaking of the so rarely combined typhoid and malarial infection, may I yet be permitted to describe the course run in a case I recently followed in Piedmont, together with the Doctors Tabasso, Farnaca, Verani, and Professor Bozzolo, of the University of Turin.

From the details given it appeared that the double infection had in all probability been caught almost contemporaneously, but in two localities diversely infected, quite distinct, and at a distance from each other. At first there was a period of real typhoid fever that began early in April and lasted three weeks, showing only mild symptoms as far as the abdomen and sensorium were concerned; these manifested themselves to a but slight degree, except during the last week, when they became more decided, and were accompanied by diarrhœa and stupor. In this period of time the disorder was believed to be and treated as simple typhoid, so much the more as the quinine in the beginning, spontaneously taken in large doses by the patient, had in no way subdued the fever, which afterwards took a steady typical course, assuming a characteristic ascending and descending curve. Only the strong shiverings which presented themselves four or five times were thought an anomalous fact; they had certainly been determined by the sporulation of the malarial parasites, that is, they had revealed the presence of the malaria already in existence, and recognised only later, after the blood had been examined. In fact, after a brief interval of apyrexia, the fever returned, but in an irregular intermittent form, with such attacks of *febris subintrans* as simulated the double tertian fever; then it became bi-quotidian, and finally quotidian, being always preceded by shiverings and accompanied by heat and perspiration, and showing the presence of the parasite in the blood. Widal's reaction also proved positive; and we also succeeded in finding the typhoid bacillus, which was isolated after Elsner's process by Dr. Farnaca, assistant to the medical clinic of Turin.

The patient had never suffered any illness before, and both the infections contemporaneously observed in his person were certainly primitive. The malarial tertian parasite was found divided into several colonies, the examination of the blood always revealing the presence of but few parasites at differen stages of growth; the quinine only succeeded in regulating the attacks, rendering them at first bi-quotidian and then quotidian, with a duration of eight, ten, twelve hours each. The disease never presented symptoms of any gravity, neither when the typhoid fever prevailed nor in the second period, which was decidedly of a malarial nature. About this latter the only remarkable fact was the resistance of the fever to quinine, even when repeatedly administered in the shape of intravenous injections. In fact, fever appeared every day from March to the end of September, when it began to decline in in-

tensity, and at last disappeared altogether. This was, perhaps, owing to the patient's having been taken to the mountains (Salberstrand, in the valley of Susa, 1,000 metres above the level of the sea), or due to the action of Baccelli's mixture, so effectual in cases of chronic malaria. The patient had some isolated attacks even later on, at long intervals, the last just before Christmas.

The tenacity of the tertian malarial infection is pretty frequent, especially when the malady is not at once attended to, as happened in the case we are speaking of. When the fever reappeared, it was at first thought to be a relapse of the typhoid. The peculiar resistance to the specific cure was in this case certainly due to the typhoid infection that had weakened the patient and exposed him to the mercy of the malarial parasite.

Section IV.

Conclusions drawn from observations at Massowha, in Italy and America.

Several conclusions may be drawn from the observations made by both the Italian medical officers at Massowha and in Italy, and the American medical officers in their encampments in the United States and at Cuba, and which we have in this paper set forth and compared:—

(1) Pathologists in hot countries, as well as in cities and camps of temperate climates, have always shown a marked tendency towards magnifying the importance of malaria, and been too reluctant to admit of the typhoid infection.

(2) The proper and accurate clinical observations, and the modern diagnostical resources tend more than ever to put things in their right place, inverting the terms.

(3) Nowadays climatic fevers and common continued fevers are no longer spoken of; nearly all the feverish disorders that come under that head are brought down to forms of typhoid, which preponderate because this infection is cosmopolitan and the inseparable companion of man. Malaria holds only the second rank, because it asserts its power in areas a good deal more restricted, and it is not an urban disease, but confines itself to limited districts in the country.

(4) One cannot speak of a real typho-malarial symbiosis but merely of a relatively rare co-existence of the two infections which may aggravate each other.

(5) The Mediterranean or Malta fever was not ascertained among the troops during the Spanish-American war, because no apposite researches were made; besides, this form belongs rather to cities than camps.

(6) The definitive solution of the problem relative to the fevers proper to hot climes will only be obtained by the following systematic plan of research: (*a*) examination of the blood with a view to discovering the malarial hæmosporidium; (*b*) the dazo reaction of the urines; (*c*) reaction against serum with artificial cultivation of the Micrococcus melitensis; (*d*) reaction against serum with artificial cultivation of the Bacillus Eberth; (*e*) application of easy and expeditious processes (like Elsner's) for obtaining the isolation from the fæces of the typhoid bacillus, and for establishing the differences between it and the Bacillum coli.

(7) Bacillum coli of different varieties, *i.e.*, of different kinds of virulence and different pathogenic properties, can give rise to gastro-enteritis, entero-colitis, dysentery, &c. There are also cases, clinically identical with typhoid infection, which are really due only to a peculiar kind of coli-bacillar infection. To these pseudo-typhoid cases, resembling typhoid fever cases, but with negative Widal's reaction and unsuccessful cultivation of typhoid bacillus, in order to ascertain the coli-bacillar infection, must be applied the serum reaction test on the Bact. coli isolated from the fæces, urines, or from the rash of the patient, after the Neüfeld's method.[1]

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[1] The method of Neühaus' modified by Neüfeld, will probably give important results on the study of the very intricate question of the various intestinal infections, both from the scientific and from the practical point of view.

The Neüfeld method for the cultivation of bacilli from the rash is the following:—After careful sterilisation of the skin, put a drop of broth for culture on the spot, which ought to be scarified so as to have a dilution of the blood in the drop deposed on the skin; the dilution is then sowed in test tubes of broth.

The conclusions will be for a coli-bacillar infection when the results of the researches are the following: (*a*) *Bacterium coli* or *coli-simile* obtained by the cultivation from the rash blood; if no rash is present, culture may be made from stools and urine of the patient; (*b*) *positive* agglutination of the Bacilli coli isolated from the patient, *negative* for the typhoid bacillus.

NOTES FROM LAGOS, WEST AFRICA.

By Henry Strahan, L.R.C.P.Lond., M.R.C.S.Eng., F.L.S.
Chief Medical Officer.

NOTE XI.—Habitat of Culex and Anopheles.

Having recently returned from a visit to various places in the hinterland of the Colony, I record one or two interesting facts bearing on the relation of malarial fever to Anopheles.

Badagry is a town situated on the lagoon and surrounded by swamps, and is infested with mosquitos especially at certain times of the year. It has, however, a good reputation as regards malaria, for it has been noted during the past three years that there is remarkably little malarial fever among the inhabitants, and when one of the few European residents is attacked, he has either had previous attacks elsewhere, or has bad occasion to visit places away from the town. The interesting point in this connection is that I was unable to find any Anopheles there among the mosquitos I collected; and the medical officers stationed there have had the same experience. There is, however, a mosquito which superficially resembles Anopheles, inasmuch as there are brownish marks on the wings. The characteristic palpi and attitude of Anopheles are, however, absent, while those of Culex are present.

Unfortunately, the specimens I sent to the British Museum arrived there damaged, but there was enough evidence to lead the authorities to believe that it is a new species of Culex, and I am endeavouring to secure fresh specimens to send for examination.

It is, however, clear that such a place as Badagry might at any time become the habitat of Anopheles, which abound in localities near. Then, no doubt, fever would be as bad there as in other towns on the coast, and as it may have been in time past. It will be of great interest to see whether a burst of malaria is preceded or accompanied by the presence of Anopheles.

A great majority of the distant towns in the hinterland which I visited were healthy and the people suffered but little from fever, but most of these places were situated on more or less elevated ground and hills, where I found no Anopheles, but myriads of Culex, the latter breeding on the granite hills and gigantic boulders which are so striking a feature of this portion of West Africa.

The small puddles in which they bred were rain-water collections either in the naturally formed depressions due to the weathering of the granite, or the artificially produced oval shallows due to the grinding of corn, &c., by the native women. But in towns situated in valleys (especially near streams, which, of course, overflow in the rainy season), I have found Anopheles abundant, and was told that the people suffered at certain times from fever, after the rains, as far as I could gather.

In the *British Medical Journal* of June 9, I see a note in which Dr. Vickerstaff, of Jamaica, directs attention to the fact that Culex breeds in the water collecting in epiphytal plants of the West Indies. This I noted many years ago, when in Jamaica, while examining the water collected in the "Wild Pines" (Tillandsias). I found numerous larvæ of Culex and other water-insects together with diatoms, &c. This I mentioned to Major Ross last year and discussed with him the possibility of Anopheles being found in such plant-collected water. I have, however, not, so far, found Anopheles in such situations.

Certain species of Culex appear to breed in moist earth where vegetation is dense but no actual pools exist. Hence the value of admitting air by removing thick undergrowth in the "bush," and allowing moisture-laden soil to dry by evaporation where draining is not necessary.

Whilst it is well known that Culex breeds in flower-vases, it is not apparently known that Anopheles may do the same. In two infested houses, where no breeding place for the larvæ of the Anopheles could be found, I directed attention to the flower-vases on the dining-tables, and in each case it was found that a copious supply of Anopheles' larvæ existed. The West African "boys" do not, if they can help it, overburden themselves with work, and are content to change the flowers, and not the water, in the vases; hence, in time, low vegetable growth may occur which nourishes the larvæ of Anopheles, while those of Culex eat the decomposing vegetable and animal matter contained in the water.

The broken half-bottles, so popular in West Africa for placing on the tops of walls to make a *chevaux de frise*, are splendid breeding places for mosquitos, but I am not sure whether Anopheles' larvæ are often found in them.

July 13, 1900.

NOTE XII.—Anchylostomiasis.

In Dr. H. A. Alford Nicholls' note on "Anchylostomiasis in the Leeward Islands," which appeared in the Journal of Tropical Medicine (for May this year), he states that in 1897, Galgey, of St. Lucia, asserted that anchylostomiasis is endemic and prevalent all over the West Indies.

When, in 1883, I discovered the hitherto unsuspected existence of the Anchylostomum duodenale (*vide British Medical Journal*, 1884 or 1885—I have not the reference at hand), being led thereto in studying the form of anæmia which prevailed, I ventured to think that eventually the parasite would be found widely spread in the whole neo-tropical region. Not many months after the publication of my observation, the existence of the worm in British Guiana was reported; in 1897 it was recorded from St. Lucia by Galgey, and in 1899 from Antigua by Dr. Macdonald. I have grounds for believing that it exists in the Isthmus of Panama, so the presumption is certainly very strong that this worm, in one or other of its species, is spread throughout the whole West Indian group and adjacent continent.

I have already, in Note III., reported its existence in Lagos, and, with the exception of extremely cold countries, it seems doubtful whether there is any place in the world which may not be the habitat of this nematode.

July 13, 1900.

A CONTRIBUTION ON THE QUESTION OF YAWS AND SYPHILIS.

By A. B. Duprey.

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There appears to be a great controversy on the subject of yaws and syphilis as to whether or not the two diseases are one and the same. Some observers give it as their emphatic opinions that the two diseases are but different phases of the one serious condition, namely, syphilis; while a few, more cautious than the others, conceal their opinions by quoting the different points of clinical interest in which the two conditions vary. Then the contagious nature of yaws is still a moot question, at least among the medical profession, for strange as it may appear, the lay public are strongly convinced of the "catching nature" of yaws. This is no doubt due to the disgusting appearance of an advanced case of the disease as it is presented to the minds of the non-calculating laymen; but when calm judgment and a critical professional mind is brought to bear on the subject, there is no reason at all why the malady, however horrible it may appear to the sight, should be thought contagious. Long before I had seen yaws I had both heard and read of its identity with syphilis, and on the very first opportunity that a case of yaws presented itself to my examination I thought "surely this is syphilis?" and a thought, I have no doubt, which would at once occur to a beginner not yet acquainted with yaws. After comparing the two diseases, however, I began to perceive the great difference that exists between the two conditions, notwithstanding the persuasion of an old practitioner, who held that yaws was but "degenerated syphilis."[1]

Mr. Hutchinson states definitely that the disease yaws is syphilis, and certainly an opinion, coming as it is from such a very high authority, ought to carry considerable weight, but at the same time his experiences, drawn chiefly from published reports and other accounts relative to yaws, should give way before those of personal experience. Whether yaws as noticed in different countries and under different names is one and the same disease, is a question far from being settled, and opinions are very much confused on the subject. That yaws is not syphilis, and, moreover, has nothing in common with it, is, I think, fairly unanimous among medical men of the West Indies who have given the subject their close study. Syphilis is indeed very common in the West Indies, and opportunities of contrasting the two diseases are often and many. Mistakes, however, do sometimes occur, especially with that variety of yaws which I would call papular yaws, and which do not arrive at the Frambœsial stage. Here is an example, a young man, aged 20, was admitted suffering from a papular eruption covering his body; the parts affected were principally his face, chest, and back. He was a muscular and well developed black with no aches or pains about him; he had come only "for the sake of the spots" on his body. He was treated for syphilis for about a month without any result. I then saw him and came to the conclusion that he was suffering from papular yaws. There was no history of any chancre or sores of any kind, there were no pains, sore throat, glandular enlargement, or any of the classical signs which would indicate that he had an attack of syphilis. A close examination of the rash with a lens brought out the fact that several of these papules all over him were tipped with amber-coloured points. The papules were discreet and irregularly scattered all over his body without the least attempt at being symmetrical. I could discover no desquamating patches such as one observes in the Frambœsial variety. The papules varied much in size but nearly all were more or less capped with the same amber-coloured material. There was not the least approach to a papular syphilide. This clinical feature of papular yaws decided me and I at once put him on simple arsenic treatment with the best results. The man so far noticed the good effect of the change that he was averse to the former treatment.

Published descriptions of yaws from other parts of the world are so much the more interesting, in that nearly all differ more or less with the West Indian disease, to wit, some suspected cases of yaws that appeared in the Journal of Tropical Medicine (Feb., 1900) by Mr. W. S. Griffith, in which I was much interested. I believe they were syphilitic in as much as syphilis is very prevalent among the Kaffirs, and that the cases did well under mercurial treatment. The manifestations of syphilis is well known to assume different guises and shapes, and it is not surprising to find a few differing in clinical features in a place where the disease abounds. I am not as yet convinced of the good effect of mercury on yaws lesions. That yaws was imported in the West Indies by the slave traffic there can be no doubt, though not simultaneously, as appears to be generally believed, as it did not make its appearance until three or four years had elapsed after the introduction of the slaves. That yaws also suddenly increased in the West Indies with the Coolie immigration from India is no less clear, a fact which seems to have escaped some acute observers. Thus the statement by Dr. Manson (tropical disease) that yaws became very prevalent in the West Indies since the Emancipation, which, he says, "has permitted the West Indian negro to revert to some extent to the state of savagery from which he had partly emerged," is absolutely untrue, and proves without a doubt how utterly ignorant Englishmen are kept regarding the conditions of these colonies. Far from going back to a state of savagery the negroes of the West Indies have steadily advanced since the Emancipation.

I have ventured to suggest a theory of the cause of yaws, such as we know it in the West Indies, which is that the disease is produced by the abuse of the commoner mangoes (*St. Mary's Hospital Gazette*, Oct., 1899). My opinion has been strengthened since I have been in the Grenadines, for this reason, that mangoes do not as yet form the staple diet of the people as in the other islands. There are, indeed, very few trees of that fruit to be found in these islands, and the almost practical absence of yaws as compared to Grenada and St. Vincent is very remarkable. On the other hand, syphilis is very

[1] *Archives of Surgery*, April, 1899.

common, so much so, indeed, that when in a doubt as to the nature of an intractable ulcer or tumour I give anti-syphilitic medicines often with beneficial results. One seldom sees a primary sore out here, because in nine cases out of ten they either remain untreated or go to the druggist, and it is only when the disease is far advanced that medical advice is sought, which no doubt explains why syphilis is so very severe in the West Indies.

I mentioned arsenic as a treatment for yaws. It is the drug which I have found answer best. Any medical man might prove its efficacy in a fresh case of yaws by suitably and gradually increased dosage. Old standing cases would, of course, require a length of treatment in proportion to the duration of the disease; and he must remember, moreover, that success in medicine is not to be gained by any show of impatience.

NOTES FROM SOUTH AFRICA.

By Major M. T. Yarr, R.A.M.C.

No. 9 General Hospital,
Bloemfontein,
Orange River Colony,
July 7, 1900.

The number of patients in this hospital at the present date, 530 only, is eminently significant of the improved state of health of Bloemfontein. Of the 530 only 400 are in the marquees or hospital proper; the rest are trivial cases, such as blistered feet, varicocele, &c., accommodated in the bell tent annexed for convalescent patients. Enteric fever still accounts for the majority of admissions, but nearly all the cases come from De Wet River and other out-stations, or are transfers from the Irish hospital, which has folded its tents and gone to Pretoria; surgical cases are few, and admissions for wounds are becoming rare. The weather is almost ideal just now—dry, warm, sunny days; cold, but not too cold nights; on most days we are enabled to raise the curtains of the marquees, and in my own division I have many of the patients carried outside the tents altogether in their beds. The more I see of enteric fever the more convinced I am of the beneficial effect of treatment under canvas; the mortality percentage (12·3 *vide* my last letter) has been unusually low, in fact, I believe, unprecedented in the field, and has been several times exceeded in epidemics amongst the civil population at home; and many apparently hopeless cases recover with a rapidity little short of miraculous. Dysentery, which at one time threatened to become epidemic, has now almost disappeared from Bloemfontein and its immediate neighbourhood; most of the cases which we still—owing to defective medical nomenclature—have to return under that heading, are cases of the harmless colitis endemic in the Orange River Colony. The rarity of rheumatic and pulmonary complaints is extraordinary, in view of the sudden fall of temperature after sun-down; judging from our experience here, the time-honoured theories as to the causation of these diseases must need revision.

Inoculation Statistics.

I am now able to furnish some statistics which may be of value when the vexed question of the efficacy of inoculation as a preventive of enteric fever is ripe for discussion. Two facts concerning these figures must be borne in mind, however, first, that in the absence of the men's documents I have had to accept patient's own statements as to whether they have undergone inoculation or not: second, that I am of course, unable to furnish the most important figures of all, viz., the comparative percentage of *attacks* of enteric fever in inoculated and non-inoculated—for this we must wait till the end of the campaign, when the Herculean task of collating the "Medical History Sheets" now at Cape Town with the "Admission and Discharge Books" of the various hospitals, must be undertaken. Where patients have not been in a condition to make a statement, I apply the word "doubtful" to the cases.

1,000 Cases of Enteric Fever.

Inoculated.	Non-Inoculated.	Doubtful.	Total Deaths.	Deaths in Inoculated.	In Non-Inoculated.	In Doubtful.
387 ..	586 ..	27 ..	123 ..	32 ..	64 ..	27

Leaving out "doubtful" cases this gives the following percentage mortalities (nearly):—

8·2 per cent. in inoculated.
10·9 ,, ,, non-inoculated.

Including and maintaining the same proportion in "doubtful" cases, the percentages are as follows (nearly):—

10·8 per cent. in inoculated.
13·3 ,, ,, non-inoculated.

"Aborted" cases of enteric fever, where the temperature falls to normal in the second week, a class of cases first described many years ago by Sir William Broadbent, are occasionally seen here, but I have not been able to satisfy myself that they occur more frequently among the inoculated than among the non-inoculated; on the whole I think they are more rare here than at home.

At the first glance the above figures would seem to indicate that inoculation has some small effect in lessening mortality; but a little consideration will show that many other factors still require to be taken into consideration before coming to a conclusion. All that can be said is that, such as they are, the figures are slightly in favour of inoculation, but they must only be considered in the light of an *interim* report. In the beginning I endeavoured to keep statistics bearing on other important aspects of the question, but was forced to give up the attempt owing to the pressure of other work—a pressure of which it is difficult to convey an adequate idea to practitioners at home.

Bilateral Typhoid Gangrene.

Cases of bilateral gangrene due to enteric fever are so very rare—only eight cases have been recorded—that the following case should be of interest:—

Pte. A., a weakly man of 23, was admitted on May 23, from De Wet River, suffering from enteric fever. The disease ran the usual course of a case of medium

severity. Some patchy congestion of the left lung appeared during the first week after his admission, but soon disappeared; the heart sounds were normal, though feeble and "slapping." By June 11 pyrexia had gone, the temperature fell below normal and remained sub-normal morning and evening. Patient was in excellent spirits and ravenously hungry, very anxious for solid food, much emaciated, no bed-sores; all through he had suffered from cold feet. He improved slightly daily; on June 18 he had custard pudding and arrowroot, in addition to fluids, and on June 21, much to his delight, he was put on stewed chicken diet. All this time, since June 11, the temperature was never higher than 97·8°, but he put on a little flesh, the tongue cleared, and diarrhœa entirely ceased; the coldness of his feet annoyed him and the night-sister placed a hot-water bottle in his bed every night. On the afternoon of June 27 he complained that his feet were colder than usual, but a hot-water bottle relieved him. Next morning, June 28, he said his right leg had pained him all night and the same afternoon Mr. Leaming, civil medical officer, in charge of the case, asked me to see him. Patient was very cheerful, thin, but not anæmic; he said the pain was quite gone but he "had no feeling in his feet." On examining the lower extremities the right was found absolutely cold, like that of a corpse, up to the junction of the middle and lower third of the thigh, without the slightest pulsation in any part; the right femoral artery could be distinctly felt as a hard, lumpy, pulseless cord between Scarpa's angle and Poupart's ligament. The left lower extremity was cold as high as the middle of the leg; a feeble pulsation could be felt in the femoral artery down to Scarpa's angle, none in Hunter's canal or below. I asked Mr. Smith (the Manchester consulting surgeon who came out with us as a temporary lieutenant, R.A.M.C.) to see the case; we agreed that operative interference was absolutely out of the question, even if it were conceivable that the patient could stand a double amputation—right hip-joint and left thigh—the evident fact that the blood-clot extended far into the right external iliac artery rendered operation unjustifiable. The poor fellow was made as comfortable as possible with cotton wool, hot bottles, &c., and given chicken, champagne, eggs and beef-tea, which he took freely. Next day, the right lower extremity was mottled with faint yellowish-green, bruise-like patches as high as the lower third of the thigh, and the left with similar but fainter patches to the middle of the leg; in the evening of the same day low muttering delirium came on; patient sank very gradually and died on June 30, three, possibly four, days after the onset of the gangrene. At the *post-mortem* examination the right femoral artery was found filled with blood-clot, which extended right up the external and common iliac to the aorta; the left was filled with clot up to within an inch of Poupart's ligament; no signs of endarteritis could be detected, but the case was evidently thrombosis, not embolism.

I have heard since of a case under the care of Mr. Bowlby, of the Portland Hospital, in which the right femoral was obliterated. Mr. Bowlby amputated at the hip joint, but the patient died, and at the necropsy the clot was found extending up to the aorta.

Two Cases of Liver Abscess.

Both of the following cases present points of great interest:—

(1) *Multiple Abscess.* — Patient, a trooper in a Colonial corps, was admitted in a very prostrate condition on June 14; he stated he had been suffering from dysentery off and on for four weeks, but had "stuck it out" until he broke down completely the day before admission. He was much emaciated; tormina and tenesmus incessant; stools stinking, rather scanty, yellowish-grey, much mucus, but not much blood. The patient's condition steadily got worse; Mr. Milton, civil medical officer, under whose care he was, tried ipecacuanha, sulphate of magnesia, opium, and finally nitrate of silver enemas (10 grs. to pint) as a *dernier ressort*. The latter afforded most relief; in fact, the patient used to ask for them. The liver dulness was increased in area but not remarkably so; there was no tenderness, and the temperature—a little over normal—presented nothing characteristic. Patient had been born and had lived all his life in Tasmania prior to coming to Africa, and had never had dysentery or other serious illness before. Death took place on June 24, ten days after admission. At the necropsy typical dysenteric ulcers were found in the colon throughout its entire length; the liver was enlarged, but not excessively so, and simply riddled with abscesses varying in size from a small pea to a plum, filled with yellow, apparently "laudable" pus. Abscesses were in the right and left lobes and lobus spigelii, on the surface, everywhere. Mr. Milton estimated the number as at least fifty. I did not hear of this case until after death—the patient was not in my division—and then unfortunately was unable to be present at the necropsy, owing to illness. The microscopic examination of stools, ulcers, and abscesses would have been full of interest, and the liver itself would have been a valuable addition to a museum of tropical medicine. I have had a pretty considerable experience of liver abscess and dysentery, but have never seen a case in which the abscesses were so numerous, nor have I ever known a case in which abscess supervened so early. I may add that Major Reade, R.A.M.C., was also present at the necropsy, and fully confirms the statement as to the number of abscesses.

(2) *Single Abscess.*—This patient was admitted in almost a moribund condition, under the care of Mr. Smith, R.A.M.C. He was sent down from De Wet river with acute dysentery, from which he had been suffering a week; he had previously suffered from the same disease in Burmah six months before. The Burmah attack had been a slight one, but he had felt "out of sorts" ever since. On admission he was in a practically hopeless state, but quite conscious; dysenteric diarrhœa was almost constant, the area of liver dulness was much enlarged and he complained of pain in the right supra-clavicular region. Next day he was no better; Mr. Smith passed an exploring needle in the eighth costal interspace between the axillary lines and found pus, but on consulting with Major Reade, R.A.M.C., considered operation unjustifiable in the patient's collapsed condition. Patient died next day, and Major Reade kindly asked me to be present at the necropsy; I had not seen the case

during life. The colon was so full of ulcers that it was impossible to find any scar tissue left by the old attack. The right lobe of the liver was converted into a huge abscess cavity containing over 30 oz. of typical liver abscess pus, chocolate-coloured, glutinous, difficult to catch in the little bottle I employed to obtain a specimen for microscopic examination. I took three slides for examination purposes from the surface and sides of ulcers, three from liver pus. The former contained no amœbæ; in the latter they were very numerous, averaging two in every field and moving sluggishly on the application of heat to the warm stage; each shewed clearly the granular endosarc and clear ectosarc described by Manson. The necropsy was made six hours after death.

That the cause of the abscesses in these cases was dysentery is, I think, undoubted; but I believe the abscess in the second case to be due to the old attack, and not to the disease contracted in Africa.

I have examined the stools, or rather the gelatinous portion of them, in nine cases of dysentery recently admitted, but could only find the amœbæ in one—the only serious case, the man being still in a somewhat grave condition. Time does not permit me at present to enter further into this question of the relation of amœbæ and dysentery, but I hope in a future communication to be able to give some interesting facts on the subject.

ON PECULIAR CONDITION OF THE HAIR.

By Irving P. Lyon, M.D.
Buffalo, N.Y., U.S.A.

Your issue of April, 1900, contained an article entitled "Peculiar Condition of the Hair," by R. C. Bennett, M.B., C.M., Government Medical Officer, Trinidad, which described and illustrated a remarkable matting and twisting of the hair of a negro child. Dr. Bennett stated that the condition had been named by some French writer "Plic" (?).

May I add a few words on this subject? The affection has been described under different names from very early times and from all parts of the world, and indeed reference to it is probably made in the Bible. The usual medical name given to it is *Plica polonica*, or Polish plait. A very thorough and interesting monograph on the subject (*Plica polonica*, graduation thesis, illustrated, pages 37, *St. Louis Medical and Surgical Journal*, December, 1897, and January, 1898) has been written by Dr. Francis E. Frouczak, of Buffalo, N.Y., treating of the disease historically as well as clinically. This writer gives a list of 174 different synonyms of the affection from 21 different languages. The affection is most common among Polish Jews, but is found widely distributed among all peoples of the lowest station of life and of uncleanly habits. In fact, the condition is simply the result of filth, and consists in the matting together and interlacing of the broken shafts of the unkempt hair, often aggravated by a process of inflammation of the underlying skin and by pediculosis. It is, therefore, not to be regarded as a distinct disease, but rather as one of the results of filth. It is mostly cured by cutting the hair and by a decent regard for cleanly habits of body. The condition is not uncommon in Buffalo among the Polish Jews, and cases of it are occasionally seen at the city dispensaries.

References to the affection, under the name of *Plica [polonica]* are found in Webster's Dictionary, and in many of the text-books of dermatology, pathology, and general medicine.

CEREBRO-SPINAL FEVER ON AN EMIGRANT SHIP.

To the Editor of "The Journal of Tropical Medicine."

Downing Street,
August 3, 1900.

Sir,—I am directed by Mr. Secretary Chamberlain to transmit to you the accompanying copy of an extract from a report by the Immigration Agent-General for British Guiana to the Governor of that Colony, having reference to an outbreak of cerebro-spinal fever on the "Clyde" while conveying immigrants from Calcutta to Georgetown, and to suggest that, if you think fit, it might be published in your Journal with a view to eliciting additional information on the subject.—I am, Sir, your obedient servant,

C. P. Lucas.

EXTRACT FROM A REPORT BY THE IMMIGRATION AGENT-GENERAL TO GOVERNOR SIR W. J. SENDALL, DATED MAY 16, 1900, Nos. 1160-1361.

I have the honour to report for the information of your Excellency that the Ship "Clyde," 1654 tons, N. G. Hatch, Master, and R. Fonseca, Surgeon-Superintendent, arrived in this port on the 9th inst. with East Indian immigrants classified as follows:—396 men, 181 women, 41 boys, 36 girls, 6 infants born at sea; 660 souls, equal to 615½ statute adults. Among the immigrants who embarked were 15 who had previously resided in British Guiana, and 21 in other colonies.

(2) There were eight births and six deaths during the voyage. The deaths were caused chiefly by cerebro-spinal fever, in regard to which the Surgeon-Superintendent made the following remark in his Report of Arrival: "This might have been a very successful voyage had cerebro-spinal fever not broken out in the ship. It is one of the most fatal diseases, and very little can be done for it as regards treatment."

(3) In his journal the surgeon made the following special report on the subject:—

"*Details of Outbreak.*—At 5 a.m. on the morning of January 27, that was, three days after embarkation, and the first day at sea, a young woman named Dhani, No. 479, was suddenly taken ill in the 'tween decks and rapidly became unconscious. She recovered consciousness in an hour and then complained of severe pain in the head with fever; the head was drawn back with frequent vomiting and delirium, and all other nervous symptoms. This woman lingered for fifty days, became insane, and died of exhaustion."

"On February 1, that is, five days after the first case, male coolie Baldeona, No. 674, was attacked. He first complained of fever and severe headache. In a few hours he became comatose and never recovered consciousness, and died in six hours after he was attacked."

On February 6 another man named Sumbar, No. 214, was attacked in the same way. He had the same symptoms; severe headache, vomiting, slight convulsive movements and fever. He recovered from the first attack of unconsciousness, could answer questions rationally at times, then deep coma set in again and he died. This case lingered four days. Then on March 19, that is, forty-one days after the last case recorded above, a man named Adhin, No. 477, was attacked with cerebro-spinal fever. He had exactly the same symptoms as the man Baldeona. The attack was sudden, fever, extreme headache, vomiting, deep coma, and death in a few hours.

"It seems strange that after being free from the disease for forty-one days that this man should be attacked. Every particle of clothing or anything likely to convey infection was thrown overboard at once, and the ship and hospital was disinfected with carbolic acid or perchloride of mercury over and over again. Another strange fact is that this man Adhin, No. 477, came to the depôt with the same batch of coolies as the woman Dhani, No. 479, who was the first case attacked with cerebro-spinal fever. This points to the period of incubation being very prolonged. All the people attacked were young, strong and healthy-looking, and in very good condition, and not weakly and malarious coolies. The temperatures were never high, mostly 101°, and very irregular, sometimes 103°. The attacks were all sudden and there were no premonitory symptoms. The constant symptoms in all these cases was the sudden unconsciousness passing into deep coma, the head fixed backwards with intense pain all over the head and neck. The delirium consisted in crying out certain words, over and over again, such as the Hindustani for 'Oh! my head!' 'My father!' 'My mother!' Wild frightened look on all the faces as if seized with some sudden terror. Convulsive movements were the exception in these cases and there was no paralysis."

"The *post-mortem* revealed acute inflammation of the membranes of the brain and spinal cord with small hæmorrhagic patches in the membranes and blood, and serum was found effused at the base of the brain. The surface of the brain and medulla looked congested."

"(2) *Meteorology.* There was nothing in the weather as far as I could see to cause this disease. In fact I never had more perfect weather on any passage. It was fine and clear with steady breezes, and with no extremes of temperature, no damp and hardly any rain; only one rough day off the Cape on the passage. The people spent the whole day in the fresh air, and were not confined at all to the 'tween decks from stress of weather. I personally do not believe that weather has anything at all to do with cerebro-spinal fever, for I have gone down the Bay of Bengal in the S.W. monsoon when the coolies have been battened down for days and never had cerebro-spinal fever in the ships, and on this passage no one could wish for better weather than we had, and, as a further proof, all the other cases treated, such as malarial fevers, pneumonias and bronchitis, although very severely attacked, made rapid and good recoveries, and not a single case was lost, and it was greatly due to the fine weather we had."

"(3) *Evidence of Contagion.*—There was not the least evidence of contagion; the cases were attacked in different parts of the ship, two aft and two forward, and all four widely apart from one another. There was no relationship or communication between these people. As soon as a case was discovered it was isolated in the hospital, and the place occupied in the 'tween decks disinfected at once with perchloride solution. None of the attendants or sick nurses were attacked."

"(4) *Sanitary Defects.*—The ship 'Clyde' is one of the best ventilated ships in the coolie service, and she was kept clean and disinfected daily. She was not overcrowded as she did not on this voyage carry her full complement of adults, and has ample room for fifty more people. No sanitary defect has caused this disease on this voyage."

"(5) *Influence of Treatment.*—All treatment is most disappointing in this disease. I have had to deal with cerebro-spinal fever on several voyages. Cold lotion to the head affords great relief, and if ice could be had I am sure it would do more good. I have followed the routine practice of applying a blister to the nape of the neck, but never found any benefit from it. Speedy relief of the bowels either by purgative or enema appears to relieve the vomiting. In treating the symptoms I find that bromide of potassium in large doses and morphia hypodermically affords most relief. I have given quinine in some form in all the cases, but never found it do good or harm; it does not influence the temperature nor relieve the headache. This is one of the most terrible diseases that one has to deal with, terminating either in death or insanity. In my experience of about thirty cases I have seen only one complete recovery."

"It would be of interest to us surgeons of coolie ships to know whether this disease is prevalent in India, or whether it is ever seen in the depôts of Calcutta."

(4) With reference to the surgeon's remark, that "If ice could be had I am sure it would do more good," I may mention that in 1886 the Surgeon-Superintendent of the ship "Allenshaw" expressed the following opinion in connection with an epidemic of cerebro-spinal fever, which occurred during the voyage of that ship, resulting in ten deaths: "One of the modern, small, inexpensive ice machines would probably have saved several lives."

PINTA.

By James Cran, M.B., C.M.
Belize, British Honduras.

A photograph of a case of Pinta is presented with this issue. Dr. J. Cran, Belize, British Honduras, sends with the photograph the following note:—

"I am sending you a photograph of a Carib woman which shows the characteristics of 'pinta' very well. My attention was called to the fact, that there are no illustrations of pinta given in any of the books, by Dr. Sandwith, in a letter in your Journal for November, 1899. I sent him a copy of the same photo, and we have had some interesting correspondence on the sub-

ject. Pinta occurs in about 60 per cent. of the adult Caribs of this colony. It also occurs amongst the other races, but not to any extent, and is extremely rare amongst Europeans. It is very well described in Manson's 'Tropical Diseases.'"

Dr. Osborne Browne, Assistant Colonial Surgeon, Saltpond, Gold Coast, sends also a note in connection with pinta.

"I have been transferred from Honduras to the Gold Coast. I have to thank the Editors for placing some of their space at my disposal *re* 'pinta'; and I will not fail to take advantage of their generosity as soon as I am prepared. Doubtless they will have heard from Dr. Sandwith again on the subject. He sent me the photo of a boy with typical pinta markings on the face, which is a rare part to be affected.

"Although it is a common disease here it does not exist to such an extent as amongst the Caribs of Honduras, and I am afraid that I shall have to alter my views of the disease, at all events, from what I have seen here."

GOUNDOU OR ANAKHRE (GROS NEZ).

Note and Photograph sent by John C. Graham, M.D.
Deli, Sumatra.

Enclosed I send you the photograph of a case of eburnation and swelling of the nasal process of the superior maxillary and nasal bones, evidently a case

similar to that referred to in Manson's "Diseases of Tropical Climates," p. 596, under the name of goundou. The left nasal ala is depressed and the left eye elevated, to make room for the enlargement.

The woman is a Malay from Sumatra. I was asked to remove the swelling for æsthetic reasons, but being an elderly lady I persuaded her that it was unnecessary. The swelling was not congenital.

UPON THE PART PLAYED BY MOSQUITOES IN THE PROPAGATION OF MALARIA. A HISTORICAL AND CRITICAL STUDY.

By George H. F. Nuttall, M.D., Ph.D.
Pathological Laboratory, Cambridge.

(*Continued from page* 307.)

In a letter dated February 22, 1899, from Calcutta, Ross, who was on the point of sailing for Europe, gave me a few details regarding his work which deserve mention. Shortly before his departure the researches in Calcutta were very much impeded by a blizzard. Ross found that Proteosoma hardly grows at all in Calcutta at a temperature below 70° Fahr. and that its growth is retarded below 80°. He stated that he had received specimens of *Anopheles claviger* from Grassi, but that "it is not the same as the two species of dapple-wing in which I cultivated crescents in Secunderabad in August, 1897." The two species to which he refers were described by him as "a large brown dapple-wing" and "a small whitish-brown dapple-wing." Ross continues, "Grassi says that the grey mosquito is *Culex pipiens*—it is evidently related, at all events. You remember that I found pigmented cells in one which had been caught biting a tertian case. Some experiments made lately have been negative with this mosquito on tertian; and the original experiment was not conclusive, as the mosquito may have bitten a proteosoma-sparrow previously." *This doubt does not apply to the experiments made at that time with dapple-winged mosquitoes on a case of tertian, as the insects were all bred from larvæ.*

Daniels (1899), who arrived in Calcutta on December 21, 1898, whither he had gone on behalf of the Royal Society to examine into the investigations conducted by Ross, reported as follows to the Royal Society in a communication dated January 23, 1899: He repeated Ross's experiments with grey mosquitoes and sparrows infected with Proteosoma. Out of forty-five mosquitoes fed on proteosoma-infected birds and examined after thirty-four hours, forty-four were found to contain "coccidia." In subsequent experiments the result was less satisfactory, owing no doubt to the low temperature prevailing at the time. Daniels found that about 75 per cent. of the insects contained "coccidia" after they had fed on proteosomal blood. "Coccidia" could not be found in control mosquitoes fed on normal blood or blood which contained Halteridia. He was able to watch the development of the parasites in the wall of the insect's intestine up to the rupture of the capsules and the liberation of the germinal threads. He was also able to find the latter in the salivary glands of mosquitoes. He states that the germinal threads are invisible in water, but that they are rendered visible by the addition of salt solution or stains. They stain with logwood or methyl-blue but not strongly. In salt solution they appear as shrivelled sickle-shaped bodies about 14-15 μ in length. On adding water or Farrant's solution they lose their shrivelled appearance and become more rounded. He doubts that they have been seen alive, owing to their transparency, and states that they show no movement.

Daniels also made some infection experiments on

birds by means of infected mosquitoes. Out of twenty-two birds that were exposed to the bites of grey mosquitoes which had previously infected themselves with Proteosoma, twelve (54 per cent.) acquired the disease. It will be remembered that Ross succeeded in infecting 79 per cent. of his birds in this way. This difference will probably be explained by the fact that Ross worked during the warm season and Daniels during the winter. It is interesting to note in this connection that the number of infected birds caught in a wild state is also smaller during the cold season. Ross had found fifteen out of 111 birds (13·5 per cent.), to be infected during the warm season, whilst Daniels only found one out of thirty birds (3·3 per cent.) infected with Proteosoma during December and January. He writes: "It is possible that in the cold season the birds have a greater power of resistance; and this is rendered more probable by the short duration of the proteosomal attack in my infected birds. Of these twelve, five died within the first week. In three, in which the Proteosoma had been very numerous, none could be found ten days after the invasion; in one in which they were very numerous none could be found on the fifth day. In the other three, very few are now found, though at first they were numerous." Owing to the cold weather the researches were much impeded, the mosquitoes refusing to bite as they do when it is warm, and the parasites developing very slowly.

Koch (September 8, 1899) reported experiments made by Pfeiffer on birds infected with Proteosoma. Sparrows and linnets were collected in various parts of the Campagna Romana and transported to Berlin. Pfeiffer was able by diluting proteosomal blood and injecting it into the breast muscle of German sparrows to reproduce the disease regularly, though in a varying degree. The period of incubation lasted usually four days, the affection not reaching its height usually before the fourteenth day. "Then, provided the disease did not run a fatal course, the symptoms began to slowly subside, and after three to four weeks the birds were again perfectly healthy." Canarybirds were found very susceptible. As the disease was confined to a sharply defined period in these birds, the birds were tested for immunity after they had been allowed to recover. This experiment was made upon twelve canaries, with the result that ten remained healthy, the two others being only slightly affected. *It is evident from this that birds recovering from proteosomal infection possess a marked immunity.* Linnets behaved like sparrows towards the parasite; crossbills exhibit a moderate degree of susceptibility; robins are readily infected but suffer little, whilst all other birds tested proved to be immune.

Though the formation of microgametes ("spermatozoa" according to Koch) was seen, no development of vermiculi, as seen in Halteridium, was observable on the slide. The development of the parasite was, however, followed in *Culex nemorosus*, this species proving to be the only one which could be brought to suck avian blood. When a period of twelve to fifteen hours had elapsed since these insects had sucked proteosomal blood, *vermiculi* could be found in its stomach, the same being similar to those of Halteridium, only longer and more slender. After forty-eight hours the vermiculi have disappeared from the lumen of the stomach and can be found beneath the epithelial layer, where they appear as "transparent, spherical bodies, which always contain a few pigment granules distributed in a circle, being rendered thereby immediately recognisable." These spherical bodies now begin to increase in size, and after six to seven days are filled with numerous crescentic bodies ("Sichelkeime") which are formed within a number of secondary spheres. These "crescentic bodies," the "sporozoites" of Grassi and "germinal rods" of Ross, when first liberated, are at times seen to hang together in radially arranged bundles. Empty capsules are next encountered, and the sporozoites gain access to the circulation of the insect. After the ninth and tenth day the sporozoites are only found within the salivary gland, chiefly in the middle lobe, where they are usually present in large numbers. Living sporozoites show no definite powers of motion. The spore-like bodies noted by Ross were also observed; their significance, was, however, not determined. *Infection experiments* with infected mosquitoes were carried out successfully in two cases. *As we see, these investigations confirm in every particular the results reported by Ross.* The developmental changes referred to are illustrated by excellent photogrammes made by Zettnow and Pfeiffer.

Observations on Halteridium.

We have noted above MacCallum's observations on the process of fertilisation in this parasite, an observation which has since been confirmed by Koch and his collaborators. Dionisi (1898) believes that the Halteridium infection in pigeons is caused by mosquitoes, as the infection occurs as a rule after the birds have moulted and are more liable to be bitten by mosquitoes.

It seems desirable to revert here to the researches of Dionisi, which were conducted in Grassi's laboratory and communicated to the Royal Academy of Medicine on May 28, 1898. Dionisi was at that time convinced of the accuracy of Bignami's hypothesis. He examined the proboscides of mosquitoes which had been collected in malarious districts, thinking that they might be found to contain the parasites. With the same object in view, he also examined the saliva and eggs of these insects. Most of the insects he examined belonged to the species *Culex pipiens*. His results were entirely negative. Impressed by the resemblance between the parasites found in the blood of birds and that of man, as well as by the similarity existing between the pathological lesions, Dionisi endeavoured to determine if the theory would apply to malarial infections in birds. He conducted his investigations on pigeons. He found that, though the young pigeons were bitten by mosquitoes, the adult birds were only bitten when they had shed their feathers. Pigeons acquire halteridium but never proteosoma-infection. He allowed pigeons whose blood contained Halteridium to be bitten by mosquitoes, and examined the contents of the latter after several hours had elapsed. He was never able to find developmental forms of the parasite within the bodies of the insects, as Ross has in the case of Proteosoma. Dionisi concluded that Halteridium was incapable of developing in the intestines of mosquitoes, but that

PINTA.

An epiphytic disease of the skin presenting irregularly pigmented patches.
Photograph sent by Dr. J. CRAN, Belize, British Honduras, Central America.

Bale and Danielsson, Ltd., London.

for infection to occur it is essential that the birds should be moulting. The latter experimentally determined fact, combined with the observation previously made, that pigeons under natural conditions only become infected at the period of moulting, led Dionisi to believe that they must become infected through the bites of infected mosquitoes. He considered that these observations supported Bignami's and disproved Manson's hypothesis. It is of interest in this connection to recall an observation made by Sacharoff (1895, p. 375), some years ago. He wrote: "It was necessary for our purpose to examine blood which contained an excess of flagellated parasites. I found such blood in young unfeathered crows taken from their nests situated in malarious districts . . These birds were very ill, and nearly all of them died in spite of their receiving sufficient food."

We may safely assume that bloodsucking insects also play the part of intermediary hosts to the hæmocytozoa discovered by Dionisi (1898) in bats. The latter found two species of parasites in bats. One of these is found in *Miniopterus Schreibersi* and *Vespertilio murinus*, and is analogous to the parasite of quartan fever in man, resembling the latter in its morphological and structural peculiarities, *i.e.*, in size, the amount and disposition of chromatin and pigment. The other form of parasite has hitherto only been found in *Vesperugo noctula*. It is unpigmented and resembles the æstivo-autumnal parasite of man. Reproduction forms of this parasite have not as yet been observed. Dionisi has recently found (1900) that the bat parasites do not develope in *Anopheles claviger* and that they are different species than the parasites affecting man.

Though the intermediary host of Halteridium has not as yet been discovered, it is hoped that it will be before long. In the meantime it seems advisable to note what has been discovered in relation to this parasite. Koch (September 8, 1899) was able to confirm the statement of Danilewsky that Halteridium occurs almost exclusively in birds of prey, climbers, songsters and pigeons. He, however, found it once in a partridge ("Rebhuhn") in East Africa. He found domestic pigeons as well as numerous species of wild pigeons to be particularly often affected, whilst the parasite is frequently encountered in sparrows, finches, jays and small birds of prey. In tropical and sub-tropical climates very few pigeons are found free of parasites (South Africa, East Africa on the coast and in the interior, Bombay). Pigeons collected in the Campagna Romana often harboured Halteridium, whilst those from Rome were free from them. Halteridium was not found in pigeons in Northern Germany, though it was present in other birds. About 50 per cent. of the sparrows caught in the Campagna contained Halteridium, whilst seventy-four sparrows caught near Berlin were unaffected. On the other hand, almost all *Fringilla cœlebs L.* and very many *Falco subbuteo L.* caught near Berlin harboured Halteridium. We have already noted elsewhere that Koch succeeded in confirming the observation made by MacCallum with regard to the fertilisation process in this parasite. The publication here referred to contains very fine microphotographs by Zettnow which illustrate the formation of microgametes, vermiculi, &c. According to Koch it would appear as if the vermiculi, in being evolved, leave detritis behind which consists chiefly of pigment, the same representing waste matter that has accumulated in the body of the growing parasite. At times some of the pigment is retained in the vermiculus, but it is usually free from it. Frosch has made the observation that the growing vermiculi develop fresh pigment within their bodies, from which it must be concluded that in the process of growth they continue to utilise hæmoglobin as food. "This observation is in so far significant, as we shall later see the vermiculi become transformed into coccidia-like spheres, which regularly contain a certain amount of pigment. The origin of this pigment would be problematical so long as one only knew the pigment-free stage of the vermiculi." The pigment contained in the coccidia-like bodies which are formed within the body of the mosquito (in other parasites) would not appear then to be entirely carried into the tissue of the insect (as Ross had supposed) by the vermiculus, but also to be formed within the body of the parasite whilst undergoing further development. The vermiculi lived several days in the moist chamber, showing slow motions, stretching and bending themselves or executing rotatory movements. In MacCallum's report it would seem as if the motions of the parasite were more active, which may be due to the different temperatures (?) at which Koch and the latter worked.[1]

[1] Koch makes the following remark in a foot-note (p. 11): "As against a hasty and entirely superfluous claim ('*Reclamation*') of Nuttall's (*Deutsche Med. Wochenschr.*, 1899, No. 8) with regard to the priority of MacCallum, I wish to clearly state that our investigations on Halteridium had been entirely concluded when MacCallum's work became known to us, consequently we could in no way have been influenced thereby. The priority of publication naturally belongs to MacCallum." I had thought to have sufficiently dealt with this question in what I had previously published. Since Koch, however, considers what I wrote "hasty and quite superfluous" (*voreilig und gänzlich überflüssig*), I feel it my duty to make the following rejoinder. Instead of expressing any further personal opinion (that would, indeed, be superfluous), I shall quote a passage from a publication of Professor W. S. Thayer, whose name is widely known to those occupying themselves with malaria. Thayer, in a brief review of recent progress in the investigation of malaria, writes as follows (May 27) with regard to the publication of Koch which I had the temerity to criticise: "Both in the reports of his studies in Africa and in a recent communication in the *Deutsche Medicinische Wochenschrift*, in which he describes his studies in Italy, Koch has detailed observations confirming much that has been done by French, Italian, American, Russian and German observers. The publications have, unfortunately, appeared in such a form as to give most readers the impression that the observations are original discoveries. They have been so regarded in many non-medical, and in some, particularly German, medical publications. It is but fair to say that Professor Koch's observations, while entitled to all the attention which is, of course, due to their distinguished author, are solely confirmatory in nature; Koch has not as yet made a single original observation in this field. Everything which he has described has been previously worked out and reported by others." Koch himself would, at any rate, have prevented any misunderstanding if he had written the following sentence in his first instead of in his last publication: "Our investigations have not brought much to light that is new. We, however, did succeed in confirming the observations of others, which had until then lacked proper connection (? N.) and supplement them in some points" . . .

(To be continued).

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THE

Journal of Tropical Medicine

AUGUST, 1900.

THE THIRD YEAR OF PUBLICATION OF "THE JOURNAL OF TROPICAL MEDICINE."

THIS issue of THE JOURNAL OF TROPICAL MEDICINE marks an era in its existence, for with it commences the third year of its publication. We have to thank our contributors and subscribers for their support, and we hope that they will continue to take interest in the publication.

The financial risks attending the issue of a new journal are proverbial; but we are happy to state that the proprietor of the JOURNAL has had no reason to regret starting a venture, which seemed, to all the "business" men he consulted before publishing, as a philanthropic work certain to spell ruin. Whilst wishing to disseminate a knowledge of Tropical Diseases, we must decline for the future to send the JOURNAL to medical men who have during the two years existence of the JOURNAL never paid a subscription of any sort. The omission is no doubt an oversight, but we would be obliged were these supporters to ask that the JOURNAL be discontinued if they do not wish to continue taking it. For the future **the Publishers inform us that they will only enter the names of Subscribers on their list when the Subscription is paid in advance.**

The chief difficulty lies in the fact that the supporters of the JOURNAL are widely scattered over the world, that they are frequently changing residence, thereby causing delay in communication, and sometimes, it is feared, non-delivery of the JOURNAL.

The Editors would be glad to have advice from their contributors and supporters as to whether the JOURNAL is meeting their desires and wants. It is for *their* benefit the JOURNAL is published, and it is for them to say what change is required, if any is necessary. The pressure on our columns of original matter is at times so great that but little room is left for extracts from current literature; but if we are thus placed it is satisfactory to note that our contemporaries largely quote from our columns, and we venture to think that as a means of focusing tropical literature the JOURNAL OF TROPICAL MEDICINE is fulfilling the purpose of its founders. Advice in this matter, however, is asked for; if given, it will be carefully considered; and the Editors will be thankful for criticisms upon their publication be they favourable or condemnatory.

BRITISH MEDICAL ASSOCIATION—THE SECTION OF TROPICAL DISEASES.

THE meeting of the British Medical Association at Ipswich during July 31 and August 1, 2 and 3, met with a success equal to any of its predecessors. The Section of Tropical Diseases celebrated its third consecutive meeting, and the doubts as to the possibility of the annual continuance of these meetings, freely expressed when the Section was first summoned, have now become a matter of mere historic interest. There was no falling off in either the interest attaching

to the meeting of the Tropical Section, in the value of the papers, or in the calibre of the discussions. It is, of course, unfortunate that so many papers were "communicated" merely, but with the supporters of the Section located in distant corners of the earth, the occurrence is unavoidable. The Section was fortunate in its President. Colonel Kenneth MacLeod, LL.D., Professor of Military Medicine, Netley, is well known as an exponent of medical and surgical work in the tropics; his contributions to medical literature are marked by that thoroughness, judgment and careful thought which command attention; and all those who have the good fortune to listen to his remarks at these meetings value them highly. The Vice-Presidents, Lieut-Colonel Oswald Baker (Ret. I.M.S.) and Major Ronald Ross (Ret. I.M.S.), and the Hon. Secretaries Dr. Guthrie Rankin and Major Andrew Duncan (Ret. I.M.S.) all contributed to make the meeting a pronounced success. It is with satisfaction that we point to the important part taken in the work of the Tropical Section by members of the Indian Medical Staff. The President, both the Vice-Presidents and one of the Hon. Secretaries, belonged to that service, and when one looks at the list of contributors of papers to the Section the Indian Medical Staff officers take a prominent place. Such well-known names as those of Lieut.-Colonel John Maitland, Major G. M. Giles, Major W. J. Buchanan, Capt. Leonard Rogers, Capt. Fearnside, Capt. James, &c., were amongst those who sent papers, and the reputation of the contributors was a guarantee of the excellence of the quality of the communications.

Quinine, as a prophylactic and curative agent in malaria, came through the ordeal of criticism triumphantly, and it is plain that the cinchona preparations are the sheet anchor of our drugs in dealing with malaria. Here and there deleterious influences were mentioned; disappointment in the use of quinine was occasionally expressed, but no other drug was seriously upheld as a therapeutic rival.

Dr. G. C. Low's and Capt. James's discoveries, rendering the bite of the mosquito the probable source of filarial infection, must be considered the most important contribution to research in tropical pathology since the Section last met. We were glad to see that Dr. Manson justly stated the claims of these two capable investigators, and henceforward their discovery of the part played by the mosquito in filariasis must be known as the Low-James or James-Low discovery.

The subject of Ankylostomiasis was advanced by the contributions of Giles, Rogers, Fearnside and Baker, and a mass of information was brought to light on the harmful, and so-called "harmless," powers of the ankylostomum.

Hepatic Abscess was dealt with by Colonel Macleod, Johnson Smith, Bassett-Smith and Cantlie. Colonel Macleod and Dr. Manson did good service to surgery by pronouncing emphatically their opinions that the operations undertaken for the treatment of this disease by surgeons in Great Britain was usually unnecessarily heroic.

The discussion on Yaws, introduced by Mr. Jonathan Hutchinson, was most instructive, as all subjects dealt with by this great clinician and original thinker always are. The members of the Section, however, were not inclined to follow Mr. Hutchinson in his opinion that Yaws is the parent of syphilis.

Amongst those who attended the meetings of the Section and took part in the discussions, in addition to those mentioned above, we observed Dr. Patrick Manson, C.M.G., F.R.S.; Lt.-Col. C. B. Maitland, I.M.S.; Major Wilson, C.M.G., D.S.O., R.A.M.C.; Major Castor, I.M.S.; Dr. Henderson, of Shanghai; Dr. Macleod, of Shanghai; Dr. Ringer, Canton, China; Dr. Harford-Battersby, Livingstone College, London; Mr. D. C. Rees, London School of Tropical Medicine; Dr. Mullick, London; Dr. Davies, Samoa, &c., &c.

Attendance at the Section of Tropical Disease has a stimulating effect upon workers in this branch of science; the earnestness of the speakers, the community of interest, the dawning knowledge in tropical pathology and parasitology, all contribute to teach those who take part in the work respect for each other and for their subject. And there is something in the fact that each

wishes to have a voice and part in the interesting departures and advances in tropical pathology which are coming to the front and enlisting the attention of the whole of the medical world. Of all the prominent figures at these meetings Dr. Manson stands out pre-eminent. Profound in knowledge, ever ready with new and carefully thought-out suggestions, deliberate of speech, and anxious for the reputation of his fellows —the "father of us all," as tropologists have come to regard him—we can only hope that for many long years to come Dr. Manson may be spared to come amongst us and guide us in our deliberations. The vote of thanks to the President, Colonel Kenneth Macleod, which closed the meeting, was well deserved and heartily accorded.

Article for Discussion.

"DO SARCOMA AND CARCINOMA EXIST AMONG NATIONS IN THE TROPICS?"

ALTHOUGH residing *just outside* the tropics, those among whom I work are precisely similar in habits, food, and climate to those a hundred and fifty miles south; and so I take it that my experience will be of service in this inquiry.

First, as to the general habits of the people. They are mostly agriculturists, living for the main part on a rice diet, which is spiced with small bits of fish, or small bits of pork, or more tasty vegetables. But, without doubt, their diet is for the most part vegetarian, and many do not touch meat for months together.

Carcinoma and sarcoma both undoubtedly exist, and run much the same course as at home; but there are, as far as I can judge, many fewer cases than at home.

During the last year (May, 1899-May, 1900) I have seen some 10,000 individual patients—a third of this number being in their homes and in villages — within a radius of twenty miles round the hospital (Changpoo, Fokien, China).

Amongst this number were the following cases:—

Eye.—Choroidal round-celled sarcoma (verified by microscope) 2
Melanotic sarcoma (verified by microscope) 1
Retro-bulbar sarcoma (not yet microscoped) 1

Tongue.—Epithelioma (typical clinically) ... 2

Œsophagus.—Carcinoma (typical clinically) ... 2

Breast.—Spheroidal-celled carcinoma (typical clinically) 3
On one I performed a Halsted, and the second was an ulcerating atrophic scirrhus, and the third a well-marked case with mass in the axilla.

Penis.—Epithelioma (typical clinically) 1
The penis and groin glands I removed most freely. Recurred in one groin after 9 months and killed the patient.

Upper jaw.—Sarcoma 2
One was a very vascular malignant growth of the alveolus which killed the patient in 3½ months. The patient was a girl of 27. The other was a typical sarcoma of the upper jaw spreading from the antrum into the temporal region, and bulging down the palate.

Lower jaw.—Sarcoma? 1
A lad of 26 with an immense ulcerating malignant growth of the left side of the lower jaw spreading deeply into the neck and utterly inoperable.

Face.—Rodent Ulcers (typical clinically) ... 2
Both typical and in the usual situations.

Back.—Sarcoma melanotic (verified by microscope) 1
Spindle-celled sarcoma (verified by microscope) 1

Besides these I have seen two cases of malignant disease of the liver and one of malignant disease of the stomach; but as to these the only evidence I have to offer is clinical, *post mortems* being impossible as yet in this region.

As far as I could ascertain, none of these patients had lived in any way a different life to their neighbours, and certainly none of them were heavy meat-eaters. Without exception they were pure Chinese. As will be seen, most of the forms of malignant disease are represented here. Malignant disease of the rectum seems very rare, and I have not been able by inquiry to learn of a single case. And this is the more remarkable, in view of the fact that chronic fistulæ and piles are extremely common, upwards of eighty having been operated upon during last year.

J. PRESTON MAXWELL, M.B., F.R.C.S.
Changpoo, Fokien, S. China.

BRITISH MEDICAL ASSOCIATION.

Section—Tropical Diseases.

Colonel Kenneth Macleod, M.D., LL.D., Professor of Medicine, Army Medical School, Netley, President, in the Chair.

Presidential Address.[1]

The Scope and Aim of the Section's Work.

Since the last meeting of this section events have occurred which have emphasised the significance and enhanced the importance of its work. The war in South Africa has stimulated into vivid reality the unity and solidarity of the British Empire. The imperial idea implies not only a community of interests—social, commercial, and political—between the mother country and her colonies and dependencies, but also a community of suffering; and as tropical conditions and tropical diseases prevail in Greater Britain to such a large extent, the study of these as they affect both the governing and the governed has come to be recognised as a matter of vital and cardinal necessity. This has been fully acknowledged by the able minister who presides over the Colonies, and who has realised more clearly than any of his predecessors the immense importance of inquiry and education as regards tropical pathology and hygiene. The Government of India has also awakened from its torpor, and taken thought and action in the same direction by encouraging research, training men in bacteriological methods, establishing laboratories, appointing commissions, and promoting special investigations.

A knowledge of the pathology and pathogenesis of disease must obviously precede and guide preventive and curative effort; and it cannot be too loudly proclaimed that this knowledge can only be obtained by systematic scientific research. The days of casual and statistical observations and dissertations have gone, and it is now universally understood that nothing will avail for the solution of pathological problems except the undistracted work of trained agents provided with ample opportunities, facilities, and appliances. The recent history of malariology—to employ a new and expressive term—is a signal illustration of the dependence of sanitary and therapeutical endeavour on pathological discovery. Laveran in 1880 furnished the key to the morbid processes which in malarious disease take place within the body by the discovery of the plasmodium malariæ. Manson and Ross pioneered the brilliant investigations which have revealed one, if not the one, means by which this organism leaves the infected subject, lives and breeds in outer nature, thus compassing the communication of what must now be admitted to be an infective disease. It is interesting to observe how this knowledge is being at the present time turned to practical account, and how it explains and gives precision to methods of prevention and cure which had previously been resorted to empirically. Koch in the Dutch Indies and German New Guinea has been attacking the plasmodium within the human host, and claims, by destroying it in that phase or stage of its existence, not only to cure the individual but to reduce greatly, or altogether abolish, the prevalence of malarious disease in the community. Sambon and Low in Italy are addressing themselves to the extrasomatic life of the parasite, and endeavouring by special contrivances and precautions to cut the morbific circuit outside the body. Koch's labours have an intimate bearing upon the subject which has been selected for discussion at this meeting, and will, no doubt, be noticed and criticised by those who take part in it. The results of the proceedings of Drs. Sambon and Low will be eagerly watched, as they will contribute an important aid to the solution of the question whether the *Anopheles* is the only medium of malarious infection, and, if so, whether this occurs invariably by inoculation.

The South African war has forced into prominent and painful attention two diseases which, although they cannot be called tropical diseases, manifest themselves with special severity as regards incidence and fatality under tropical conditions—namely, enteric fever and dysentery. The theatre of the war—the uplands of South Africa—cannot be classed, either as regards position or physical characters, as tropical. Malarious diseases, the special and predominant product and index of tropical countries, is conspicuous by its absence among the causes of disability and death occurring in the British army in South Africa. But the meteorological conditions which obtained during the early months of the campaign were most aptly described by the term "tropical." Sunstroke and sun fever were very common. I have endeavoured to ascertain whether these cases were all or mostly cases of heat shock, and whether any considerable number of them presented the phenomena of heat fever—so-called "siriasis"—and, if so, whether these occurred in epidemics or appeared to be communicable. I regret to say that I have not succeeded in obtaining any information on these points. The cases which I have met with at Netley have presented a similar history and similar sequelæ to those received from India. Perhaps, when the medical history of the campaign comes to be compiled, some facts bearing on these questions may be forthcoming. I allude to the matter here and now in the hope of eliciting information.

That enteric fever existed in South Africa and was apt to prevail in South African towns and cantonments during the summer months was well known, and its appearance among the troops engaged in this war was fully anticipated; but the excessive prevalence of the disease in a country and climate with a reputation for exceptional healthiness has come as an unpleasant surprise. No doubt the circumstances and exigencies of warfare are mainly responsible for the heavy tribute of sickness and death which enteric fever has levied. War shares with famine the malignant power of enhancing the susceptibility to whatever infection happens to be present at the place and time.

Enteric fever has been in grim evidence during recent wars on the Indian Frontier and in the Egyptian Soudan; but malaria, cholera, yellow fever, and dysentery have on other occasions been stimulated into disastrous activity by war. So with famine; malarious disease, small-pox, diarrhœa, dysentery,

[1] Delivered at the opening of the Section of Tropical Diseases at the Annual Meeting of the British Medical Association at Ipswich, July-August, 1900.

and relapsing fever have attended or followed it, and at the present time cholera and plague are raging among the famine stricken in India. The infection of enteric fever seems to be ubiquitous, portable, and peculiarly facile and subtle; and perhaps the most urgent question of the hour is how to mitigate its prevalence in the British army, in which in times of peace it causes one-third of the total mortality.

It is important to note that a very marked contrast exists between the ordinary incidence and mortality of the disease in temperate and tropical or sub-tropical countries—in England and Canada on the one hand, and in India and Egypt on the other. A similar contrast appears in the French army stationed in France and in Northern Africa. How much of this great excess is due to tropical conditions, topical and climatic, and how much to remediable sanitary defects, it is not easy to say.

But, side by side with the excessive suffering of the army in India, we are confronted with the remarkable fact of the immunity of the native population. Whether a similar immunity exists among indigenous races and habitual residents in South Africa is an interesting question. Evidence seems to indicate that it is so. The native immunity in India, though not absolute, is undoubted; its cause has not been satisfactorily ascertained. It has been attributed to habituation to minute dosage of the contagium, to protection conferred by attack during infancy and childhood, and to racial resistance acquired in the course of generations through both these influences. Some experiments by Freyer and others indicate that natives give positive reactions to Widal's test; but more extended and exact investigations on this point are desirable. It is quite certain that the immunity is not due to superior sanitary conditions. Whether a similar immunity—temporary or permanent—can be engendered in European subjects by a process of inoculation such as has been devised by Professor Wright, of Netley, and practised on a large scale among soldiers proceeding to the seat of war, is a question, the reply to which is awaited with eager anxiety. Some figures obtained from Ladysmith have been published by Professor Wright, which seem to show that some immunity is conferred by these inoculations, but though encouraging, they are by no means demonstrative. Similar procedures for creating an immunity against cholera and plague, initiated by Professor Haffkine, have in India been attended with satisfactory results. But although a certain measure of preventive success has been obtained by these inoculations, the employment of them appears at present to be practicable and useful only as an emergent expedient in the presence of a serious outbreak; and the prevention of cholera, plague and enteric fever on a large scale must apparently be essayed on other lines and by other methods.

Dysentery has been very rife in the South African army, but the disease has exhibited mostly a mild type and been amenable to treatment. In some camps it has presented the aspect of an epidemic or infectious disease; but whether the infectiousness is apparent or real—due to common exposure to certain noxious conditions or to communication from man to man of some specific contagium—it is impossible in the absence of knowledge regarding the nature of the contagium or contagia of dysentery to say. The dysentery of war and famine is believed to be infectious, but notwithstanding much able and laborious research we have yet much to learn concerning the pathology and causation of dysentery. Imperfect conservancy, foul water, alternations of temperature, exposure, fatigue, and bad food, which are undoubtedly adjuvants if not factors of dysentery, have been in baneful operation in this war, and a new disease resembling dysentery has been described under the name of "dust colic." This seems to be a muco-enteritis caused by the swallowing with water and food of irritating particles of grit blown about by dust storms. The presence of the grit in the evacuations does not seem to have been sought for or found.

The persistence of plague in India and the appearance of the disease for the first time south of the equator—in Mauritius, South Africa, South America, and Australia—are events deserving of special notice. The disease has during its present prevalence confined itself mostly to warm and hot countries, and, though not exclusively a tropical disease, nor apt in the tropics to be at its worst when conditions are most typically tropical, it appears to find in tropical countries and circumstances the most favouring environment. It is curious to remark that, while in India natives appear to be readily susceptible to the infection of plague, Europeans, though not absolutely insusceptible, exhibit a comparative immunity—the reverse of what happens as regards enteric fever. This immunity is doubtless what I venture to call a sanitary immunity, due to a purer personal domestic and social life, and perhaps to circumstances and habits rendering admission of infection less easy. This kind of immunity is also observable in some places—in Calcutta, for example—as regards cholera. How far an immunity of this sort is capable of being achieved as regards the infection of enteric fever, it is not easy to say. Certainly it has not been accomplished as yet in India or Egypt. The Bermudas used to render the highest ratios of enteric prevalence and mortality, but within recent years considerable reduction of these rates has occurred through sanitary reforms in the matter specially of water conservancy and sewage disposal. Similar causes have reduced the burden of enteric suffering in the French army of Algeria, and the power of sanitation has also obtained signal illustration in the banishment of beri-beri from the Japanese navy. Great Britain appears also to have acquired an immunity against cholera through sanitary reform and effort. These experiences are full of encouragement and hope.

I trust that I have succeeded by these discursive observations in showing that recent events have expanded the scope and aim of our work in this Section; and it seems to me fitting that we should at the commencement of our sectional labours remind ourselves of what these are. Our concern is not only with exclusively tropical diseases, many of them strangely named and imperfectly investigated and understood, which may be encountered and contracted in hot places where Europeans are compelled to reside for purposes of protection, administration, or commerce, and are not as a rule met with outside of the

tropics. More important are those diseases, originally or essentially tropical, which may be disseminated by intercourse with the tropics, and may prevail for a time in extra-tropical localities in which they are not habitually present. And, finally, there are the diseases which are not specially tropical, but which are liable to be aggravated in prevalence or severity by tropical conditions. These three classes represent a wide field of research, and, in addition, interesting questions arise as regards diseases which, common elsewhere, are rare or unknown in tropical countries.

The field of study thus presented has its scientific and humanitarian aspects, and its cultivation has become an essential part of the business of imperial administration. It embraces not only acute infections and the sequelæ or constitutional incapacities resulting therefrom, but includes also those conditions affecting health and life which are vaguely designated as climatic, remediable only by adaptation, or which arise from sanitary defects or neglects capable of more easy amelioration. Hygienic improvements, personal, domestic, and social, have undoubtedly raised the standard of health and the value of life in the tropics, and residence and service in hot countries offer fewer and less formidable risks than they did in times past. But behind the question of individual impunity looms the question of colonisation or the continued vigour and vitality of the race when transplanted from temperate to torrid zones. The solution of these weighty problems constitutes the reason and purpose of our distinct sectional existence.

Quinine: its Action and Value.

(*a*) *The Action and Modes of Employment of Quinine in Malaria.* Dr. Andrew Duncan (Major Ret. I.M.S.) read a paper upon this subject and opened a discussion upon the use of quinine as a prophylactic and as a curative agent. In the hands of French physicians quinine is found to be of some prophylactic value in the milder forms of malarial fever, but in pernicious varieties it is well-nigh useless. The general conclusions arrived at by Russian and Austrian military surgeons are that quinine has but little, if any, value as a prophylactic. In America Dr. Bryan states that cinchona preparations have a markedly prophylactic action. The late Surgeon-Major Parke, of the Stanley expedition in search of Livingstone, gave the officers four grains of quinine daily for ten days before entering the mouth of the Congo. During a subsequent journey of 350 miles through one of the most unhealthy regions of the world only two officers contracted fever. Dr. Duncan's own experiences in India afford support to the belief that the giving quinine is a preventive to malaria. In 1896, of fifty men of the 2nd Goorkha Rifles who took three grains quinine daily none had fever, whereas amongst the men who took no drugs 6·5 per cent. had malaria; in 1897 the same experiment was tried again, when the results were no malaria amongst the quinine takers and 9·8 per cent. amongst the non-quinine takers. During the Malay war the prophylactic benefits of quinine were not marked. West African experiences are varied. Harvey found that the sailors who took quinine had as much fever as the men who did not take it. During the Ashanti wars of 1893 and 1896 quinine as a prophylactic proved of no benefit. An enquiry in connection with this subject was promoted by Mr. Chamberlain and Dr. Manson in West Africa. The benefit of quinine as elicited by this enquiry seems to be pronounced, for in 87·7 per cent. of those who used it as prophylactic it was efficacious.

Dr. Duncan then reviewed the action of several reputed anti-malarial drugs. Arsenic as a prophylactic affords conflicting evidence. In Italy it has been moderately successful, in India it has proved very disappointing. The most eminently successful experiment seems to have been made by Dr. Ralph Leslie in the Congo Free State, where arsenic was administered during fifteen days every six weeks, and everyone who took it was rendered immune to fever. Narcotine seems of little value as a prophylactic. As a cure for malarial attacks quinine gives by far the best results, only 2·05 per cent. failures. Next to quinine as a curative comes nim bark in doses of one drachm thrice daily; failures 18 per cent. Berberis had 50 per cent. failures. Narcotine, kreat and inderjas seem to be unreliable as either prophylactic or curative agents.

(*b*) *The Prophylactic Issue of Quinine. A synopsis of an experiment on a large scale in Indian Jails.* By Major W. J. Buchanan, I.M.S. At six of the large jails of India the prophylactic issue of quinine has been tried for five years past. In some instances the results have been tested by control experiments. At the Mymensingh jail the result was "diminished fever;" at the Rajahmundri jail, one of the most malarial jails in India, quinine as a prophylactic was useful in those who had not been previously exposed to malaria; those who contracted fever had it more mildly, the duration was shorter and the recurrences fewer when the drug had been taken previously as a preventive. At the Rajshaye jail Lt.-Col. French reported strongly in favour of quinine as a prophylactic during 1896 and 1897, but he changed his opinion in consequence of the experiences gained during 1898. At the Bankura jail the prophylactic issue of quinine had distinctly beneficial results; at the Huzaribagh jail, Major Maynard gave it as his opinion that cinchonidine given in 6-grain doses daily was useful as a prophylactic, and as a beneficial agent should attacks of fever come on afterwards. Major Buchanan's experience at the Bhagalpur jail proved negative, the quinine takers and those who had not taken the drug being equally affected. Major Buchanan has never seen hæmoglobinuria or other evil effects from taking quinine.

(*c*) *The Administration of Quinine, with special reference to the Practice on the West Coast of Africa,* was the title of a paper communicated by Dr. Fielding Ould (W. Africa). Dr. Ould states that since Koch's statements about quinine the Europeans on the West Coast entertain, many of them, a dread of the drug. Quinine acts by staying the development of the malarial amœbulæ; it may do so by binding the oxygen to the hæmoglobin more closely and thus depriving the parasite of the oxygen necessary for its growth. Quinine can be in no sense a preventive, it can only deal with the parasite when it exists in certain stages in the blood, it cannot prevent the

entrance of the parasite into the blood. He quotes an experiment of Bignami's in which blood rich in parasites, but impregnated with quinine, was incapable of reproducing malaria when injected into a healthy person. Of the methods of administration that by the mouth is perhaps preferable, unless gastric catarrh is present when it may be given by the rectum or hypodermically. The prophylactic use of quinine is in many instances harmful by upsetting digestion; and it is persons who suffer from gastric catarrh or hyperæmia of the liver who are wont to be most severely attacked by malaria; in them the pernicious forms of malaria, the bilious remittent form and hæmoglobinuria, are apt to develop.

Discussion on the three papers relating to Quinine.

(1) Dr. Patrick Manson, C.M.G., F.R.S. The reputed prophylactic action of quinine is but a phase of its therapeutic action; it is the application of the drug to the parasite and not an immunising of the body against the parasite that characterises the action of quinine. Just as some varieties of the parasite are highly amenable to the drug given therapeutically, similarly its prophylactic power will be greater against such. Dr. Manson recommended that future experiments in prophylaxis be made with the aid of the microscope, and in reference to the particular type of malarial parasite it is used against.

(2) Lt.-Col. Marsden, I.M.S., stated that he had invariably given quinine, both as a prophylactic and as a therapeutic agent, for the last twenty years, and the results have nearly always been satisfactory. Lt.-Col. Marsden described the only case in which he had seen quinine cause hæmoglobinuria as that of a German missionary, who every time he took quinine declared that hæmoglobinuria supervened, and when the drug was administered by Lt.-Col. Marsden the urinary trouble developed.

(3) Mr. D. C. Rees (London School of Tropical Medicine) gave his experience of quinine as a prophylactic in Nigeria. Mr. Rees came to the conclusion that 5 grains administered daily, although it does not reduce markedly the number of attacks of fever, tends to lessen the severity and also the case mortality. He advocated introducing the needle of the hypodermic syringe into the muscles when administering quinine by this method; when introduced beneath the skin only, suppuration was apt to follow.

(4) Dr. C. F. Harford-Battersby (London) believes strongly in the prophylactic value of quinine. Although quinine might cause hæmoglobinuria, he did not believe it could cause hæmoglobinuric fever. Dr. Battersby holds that there are many minor ailments attributable to malaria, such as vomiting, neuralgia, &c., which quinine will relieve.

(5) Major Wilson, R.A.M.C., did not find in the Ashanti Expedition of 1895-6, nor on the Sierra Leone Coast, that quinine had much prophylactic value. He had used the hypodermic injection of quinine in many instances without causing local inflammation.

(6) Dr. B. S. Ringer (Canton, China) described a case of quinine (in malarial) blindness, which, however, disappeared by treatment with x.-grain doses of potassium iodide.

(7) Lt.-Col. B. C. Maitland, I.M.S., asked for information concerning the use of methylene-blue in malarial fever. During an outbreak of malaria he gave it in alternate cases with quinine and found methylene-blue gave the better results. Lt.-Col. Maitland found little danger in giving quinine in pregnancy, and he has administered it at any and every stage of pregnancy without inducing abortion.

(8) Mr. James Cantlie (London), related a case of fever in a child of 4 months of age. The child was born in England, whilst the parents were at home on a holiday from China. The fever continued for six weeks, and it was only when the mother (who was nursing the child) and the child took quinine that the fever disappeared.

(9) Dr. Henderson (Shanghai) stated that the benign tertian was the usual malarial parasite met with in Shanghai practice and that it readily yields to quinine. He believes quinine to be a decidedly dangerous drug in pregnancy, and he had seen miscarriages traceable to its administration. With opium, or better still with chlorodyne, some of the danger of quinine to pregnant women is possibly minimised.

(10) Major Ronald Ross (Ret. I.M.S.), Liverpool, drew attention to the circumstance that, in old cases of malaria, there may be a secondary form of fever due to enlargement of the liver and spleen, not directly due to the presence of parasites and *not* amenable to quinine. Major Ross advocates the exhibition of quinine for three months after fever, and believes the best form of administration is in solution by mouth.

(11) Dr. Guthrie Rankin (London) described a case of hæmoglobinuric fever developing in about fourteen months after settlement in Central Africa. The patient returned to England, had a mild attack of hæmoglobinuria, and after four months went back to Africa. During his second spell of residence there, extending over two-and-a-half years, he took quinine daily in teaspoonful doses and never had a return of malaria fever or of hæmoglobinuria.

(12) Lieut.-Col. Oswald Baker (Ret. I.M.S.), London, believes the reason of quinine failing as a prophylactic is that it is not given in sufficient doses. He is of opinion that the prophylactic dose should be the same as the curative dose.

(13) Colonel K. MacLeod (Ret. I.M.S.), Netley, pointed out the importance of using the microscope during the administration of quinine as a guide and check. Koch points out that malarial parasites may exist in the blood without causing pyrexia, and it is necessary to ascertain the infection of the community before coming to a conclusion as regards the prophylactic use of quinine. Col. MacLeod referred to the distinction to be drawn between hæmoglobinuria and hæmoglobinuric fever. The former was caused by many drugs, but the latter seemed to be a specific disease.

Filariasis.

(*a*) "*Notes on the Etiology of Filariasis.*" *By Lt.-Col. J. Maitland, Professor of Surgery, Madras.* Lt.-Col. Maitland referred to the observations made by Dr. G. C. Low concerning the presence of the filiarial parasite in the proboscis of the mosquito, and the probability of the infection of the human being by the bite of the mosquito. He regards the

evidence in favour of this theory as presumptive only and opposed to well attested evidence. He drew attention to the extraordinary immunity of Europeans to filariasis in districts where the natives were extensively infected, and regarded the fact that Europeans boiled or filtered their drinking water as an argument in favour of water being the medium of infection in natives. Lt.-Col. Maitland considers it quite possible that the young filaria may pass from the mosquito's proboscis into water instead of directly into the blood.

(*b*) *On the Metamorphosis of the Filaria Sanguinis Hominis in Mosquitoes, especially with reference to its Metamorphosis in the Anopheles Rossii and other Mosquitoes in the Anopheles Genus. By Captain S. P. James, I.M.S.* Attention was drawn in this paper to the difference in time required for the metamorphosis of the filaria in mosquitoes observed by Bancroft and Manson, and it would appear that the period of such metamorphosis is not yet determined definitely. In Bancroft's experiments and in those undertaken by Captain James, the female culex was the insect employed. They were bred from larvæ, placed under the mosquito curtains of a filariated man's bed, and when caught transferred to bottles in which ripe bananas were hung. Against the water-borne theory of infection Captain James advances the argument that the filaria die in two-and-a-half hours in pure water; and therefore too short a time of existence is possible to ensure continuance of the species. Captain James favours the idea that the filaria is carried to the human being by the bite of either the anopheles or culex.

Discussion.

(1) Dr. Manson wishes to make it known that the discoveries of Dr. Low and Captain James were made independently of each other, and that both observers are entitled to have their names associated with the establishment of the fact that the filarial worm can find exit by way of the proboscis of the mosquito. He regards the relative immunity of Europeans to filarial infection as due to the use of mosquito nets and their better sanitary surroundings compared with poor natives. The process of rendering a human being richly infected by filaria probably involved a considerable time; the individual required possibly to be bitten many scores of times by filarial infected mosquitoes before the embryos appeared in sufficient numbers to cause pathologically lesions of any clinical magnitude.

Dr. Manson drew attention to the analogy between filariasis and malaria. In both diseases a parasite is removed from the human blood by the mosquito; they both develope in the mosquito tissues; both probably quit the mosquito *via* the proboscis; both are inoculated by the mosquito bite; both exhibit a remarkable periodicity in the human blood, and both give rise to recurring fevers.

(2) Mr. D. C. Rees said that, although Dr. Bancroft (Australia) and Captain James seemed disinclined to believe that the Filaria nocturna casts its sheath in the mosquito's stomach, he thinks that it does do so. Several specimens in his possession show that ecdysis has taken place, for the filaria and its sheath can be seen side by side.

(3) Major Ross referred to the case of an Englishman living in the West Indies who suffers from elephantiasis which he ascribes to the bite of a mosquito.

Hot-Weather Diarrhœa in India. By Major W. J. Buchanan, I.M.S. The object of this paper is to draw attention to a very severe form of diarrhœa which is not cholera, but in many cases closely resembling it. Major Buchanan, after describing the sudden onset, the watery stools, the collapse and the cyanotic appearance of the patient, occasional suppression of urine, &c., thinks the disease bears the same relation to cholera in India as does summer diarrhœa to cholera nostras in England. He suggests the name thermic diarrhœa for the complaint. The disease, especially in old people, is occasionally fatal. The treatment consists in boiled milk, brandy, and the tinct. chloroformi et morphinæ co. B.P. As the diarrhœa subsides, a dose of castor oil hastens recovery.

Colonel Macleod remarked that Major Buchanan's paper raised two questions—first as to the distinction between cholera nostras and cholera Asiatica, and, secondly, what the organisms or toxins are which give rise to cholera nostras. The form of diarrhœa mentioned is very common amongst children in India.

A Note on a Case of Blackwater Fever, with Specimens, was contributed by Dr. George Thin (London). The patient from which the notes were taken died of blackwater fever in Central Africa. Specimens were forwarded to Dr. Thin by Dr. Douglas Gray for examination. Dr. Thin's conclusions are that the patient died of acute malarial fever, in which the hæmoglobinuria was a concomitant symptom. The presence, condition, and extent of the pigment in the liver and spleen showed the recent existence of the parasite in great numbers at some point of the blood circulation.

Dr. Manson remarked that, although he inclined to the belief that hæmoglobinuric fever is an expression of malarial fever, he did not think we are justified in jumping at such a conclusion merely because the malaral parasite is found in a hæmoglobinuric patient's blood. It would be strange were *post mortem* evidence of malarial infection not found in persons dwelling in so highly malarious a country as British Central Africa, whatever the cause of death.

Mr. D. C. Rees stated that he had examined specimens from the same patient upon which Dr. Thin had reported and he had come to an exactly opposite conclusion to that arrived at by Dr. Thin.

The Diseases of Goorkhas. By Dr. Andrew Duncan (Major I.M.S., retired). Malarial fever of an intractable form seemed to prevail amongst the soldiers of the Goorkha regiments he had been quartered with. He found administration of quinine by the rectum was more efficacious than by the mouth. Phthisis seemed also to run a peculiar course, passing from mild symptoms to a severe and suddenly fatal form. Mumps, measles, and ophthalmia were prevalent. Enteric fever, contrary to popular belief, is not a common disease amongst Goorkhas, in fact he had only seen one case. In reply to Mr. Cantlie, Dr. Duncan stated that he had never seen scarlet fever amongst Goorkhas.

ANKYLOSTOMIASIS.

(*a*) Major G. M. Giles, I.M.S., opened the discussion on ankylostomiasis by communicating a paper in which he referred to: (1) The history of our knowledge of the disease. (2) To his own researches on the life history of the free stage of ankylostomum, by which he established the fact that the rhabdites can live and multiply free in fæcal matter. In other words, that it is a case of heterogenesis or dimorphism. (3) Major Giles dealt with Dr. Sonsino's criticisms as regards the accuracy of his observations. Dr. Sonsino holds that the organisms described by Major Giles are Rhabditis terricola or other free nematodes, and that the appearances were really the free form of Rhabdonema intestinale. To these criticisms Major Giles gave a distinct negative as to their potency. (4) Major Giles next discussed the question of the harmful *versus* the harmless nature of the ankylostomum. He holds that the parasite is responsible for a formidable mortality, and of a great amount of chronic sickness.

(*b*) Capt. C. F. Fearnside, I.M.S., contributed a paper on *Ankylostoma Duodenale*. Capt. Fearnside related a systematic search for the ova of the ankylostomum parasite amongst the convicts and new arrivals in the central prison at Rajahmundry in India. He found that, of 678 new arrivals at the prison, 462 or 68·1 per cent. harboured the parasite. In another group of observations Capt. Fearnside found 72 per cent. infested, yet they remained in good health. About 35 per cent. harbour the ankylostomum and Ascaris lumbricoides simultaneously. At 105 *post-mortem* examinations at the jail 74·3 per cent. revealed the presence of the ankylostomum. Capt. Fearnside holds that *post-mortem* evidence goes to show that the effects of the ankylostoma are for the most part secondary and not primary, and that it is only when the anchylostoma attack a person already weakened by malaria or other illness that ankylostomiasis develops.

(*c*) *The Classification and Differential Diagnosis of Ankylostomiasis, with special reference to the type of the Anæmia*, was the title of a paper communicated by Dr. Leonard Rogers, I.M.S. The question of the relation of the kala-azar to ankylostomiasis was discussed by Capt. Rogers, and he agrees with Major Ross that kala-azar has a malaria rather than an ankylostomian basis. He regards ankylostomiasis as a disease characterised by anæmia, produced by long-continued small losses of blood through the gastro-intestinal mucous membrane, caused by the presence of several hundreds of ankylostoma acting for many months, or by still larger numbers acting for a shorter time.

(*d*) *Ankylostomiasis in Burmah* was dealt with by Lieut.-Col. Oswald Baker (retired I.M.S.).—He described the condition induced by the ankylostoma in severe form as being practically identical with that of a person bled to the verge of death. He established the fact that the ankylostomum was extremely prevalent throughout the province, and considers the prevalence of ankylostomiasis to be directly and indirectly responsible for a high mortality. He attributes the wide distribution of the disease to be due to the habits and customs of the Burmese, especially as regards their mode of eating their food.

Dr. Manson remarked that he was surprised the Rhabdonema intestinale was so seldom found by Major Giles in Assam, considering that it is so frequently associated elsewhere with the ankylostomum, and that the climatic and other physical conditions closely resemble those in Cochin-China, where the parasite is extremely common. Dr. Manson had twice encountered ankylostoma in the stools of Englishmen who had returned to this country from the West Indies.

Dr. Manson disagreed with a remark made by Dr. Fearnside in regard to the inefficacy of thymol as an anthelmintic and stated that thymol rarely fails if given in adequate and rapidly repeated doses. He referred also to the virtues claimed for betel nut by the natives of Assam, Burmah and Malaya as a prophylactic against ankylostomum. Dr. Manson believes that the parasite is generally acquired by way of the food.

(2) Major Ross remarked that he carefully followed Major Giles' investigations in the rhabditiform stage of the parasite and considers his conclusions sound. He regards the well-known fact that the disease is present amongst earth-workers as an argument in favour of Major Giles' statements. He scouted the idea that the ankylostoma parasite was harmless, and entered a plea for the much more general use of the microscope for the detection of the ova in localities where the worm is prevalent.

(3) Mr. Cantlie stated that, stimulated by the researches of Dr. Walker in Borneo, concerning the association of the ankylostomum with beri-beri, he had carried out prolonged examinations in Hong-Kong but failed to find the parasite in cases of beri-beri.

TROPICAL ABSCESS OF THE LIVER.

(*a*) *The Management of Lung Lesion consequent on Liver Abscess* was the subject of a paper by Colonel Kenneth MacLeod (Netley).—The frequency of liver abscess pointing in the direction of the thorax and opening either into the pleural cavity or into the lung was referred to. When such an event occurs a free opening into the pleural cavity must be made with resection of a rib if necessary. He pointed out the futility of operating upon an abscess of the liver after expectoration of liver pus. In all such cases the condition ought to be approached by way of the chest, and the lung treated as the offending organ; for, very soon after the pus has found evacuation by way of a bronchus, the liver abscess commences to heal and is not infrequently healed altogther, the patient succumbing to the lung lesion. Colonel MacLeod advocates the liver being left to take care of itself in such cases and the treatment confined to the relief of the lung.

(*b*) *Subhepatic Abscess* was the title of a paper read by Mr. James Cantlie (London).—By subhepatic abscess is meant a collection of pus between the capsule of the liver and the under surface of the liver substance. The abscess tends to point in the epigastrium and is probably due to a lymphangitis, and is not associated with either dysentery or any other pronounced intestinal lesion. He had been able to verify the existence of subhepatic abscess in a case in which laparotomy was performed, and he had seen

two or three cases in which the clinical evidence was pronounced.

(*c*) *The Diagnosis and Surgical Treatment of Tropical Liver Abscess.* By W. Johnson Smith, F.R.C.S. (Seamen's Hospital Society, Greenwich). In regard to surgical treatment, Mr. Johnson Smith prefers exploratory puncture to laparatomy as a means of diagnosis. He has met with almost invariable success by puncturing the liver, and has never seen any untoward effects from it. Laparotomy under the most favourable circumstances has certain inconveniences and after troubles when employed as a means of diagnosis. When treating the abscess by transpleural incision, there is the great danger of opening the cavity of the pleura. When the diaphragm is so pressed upwards as to bulge into the wound, the pleural layers may be so approximated that air does not gain entrance to the cavity—a most fortunate circumstance.

(*d*) *Abscess of the Left Lobe of the Liver, with remarks particularly referring to its amœbic causation.* By *Staff-Surgeon B. W. Bassett Smith, R.N.* (Haslar). The abscess in this instance occurred in a man in England who had not been abroad for two years. The hepatic abscess followed quickly after an attack of dysentery. Amœbæ coli were abundantly found in the pus, to the exclusion of pyogenic bacteria. Owing to the existence of the pus in the left lobe of the liver, the abscess was not found by exploratory puncture until quite late in the illness. *Post mortem* an abscess was found in the right lung, not directly communicating with the liver abscess cavity.

Discussion.

(1) Dr. Manson referred to the heroic nature of the operation for liver abscess practised by most surgeons in England, and thought that by the trocar and cannula more satisfactory results were obtained. He did not regard the presence of the Amœbæ coli in the sputum as an indication of pus in the liver; he had never met with it. Dr. Manson said he failed to understand the cause of the serious hæmorrhage met with during hepatic puncture by Bombay surgeons.

(2) Major Ross had seen a case of hepatic abscess in Liverpool where leucocythemia, but no evidence of malaria, was present.

(3) Mr. Cantlie said he had followed Dr. Manson's plan of treating liver abscess by the trocar and cannula and the insertion of a large drainage tube into the cavity with success. He stated that he had studied the dangers from hæmorrhage during hepatic exploration, and had from frozen sections of the body come to the conclusion that in a chest of 32 inches circumference it was not safe to penetrate further into the liver, in a vertical direction from the surface, than $3\frac{3}{4}$ inches.

(4) Colonel Macleod agreed with Dr. Manson that the plan followed by most surgeons was too severe. He mentioned a case in which, after laparotomy, the surgeon failed to find pus in the liver, yet the abscess burst into the bowel within three days after the operation. The transthoracic incision with removal of a rib and stitching of parts together was an unnecessary proceeding unless the pus was actually in the pleural cavity.

The Cyprus Sphalangi.

The Cyprus Sphalangi and its Connection with Anthrax was the subject of a paper communicated by Dr. George A. Williamson, of Cyprus. The so-called sphalangi is an insect resembling an ant, belonging to a genus of stingbearing Hymenoptera called Mutilla; the bite of this insect is considered in Cyprus to be the means of carrying anthrax from animals dead of the disease to human beings. After stinging, the local symptoms are a circumscribed induration and wide spread œdema with toxic general symptoms; the discharge from the puncture was found to swarm with Anthrax bacilli. The treatment consists in destruction of the indurated tissue by the cautery and hypodermic injections around the part of a solution of mercuric chloride and iodide of potassium.

Some Suggestions for the Improvement of Sanitary and Medical Practice in the Tropics was the subject of a paper by Major Ronald Ross (ret. I.M.S.), Liverpool. Major Ross dealt with the necessity for instruction in animal parasitology and comparative parasitology for medical men about to proceed to the Tropics. He advocated (1) the formation of small up-to-date libraries in the large towns in our colonies, a better supply of microscopes; (2) the necessity for improved municipal sanitary regulations; (3) the desirability of securing organisation in the matter of research; (4) the importance of establishing a central scientific authority.

Dr. Mullick (London) said there was urgent necessity for the appointment of specially-trained investigators to fill the chairs in the medical schools of India.

Colonel Macleod pointed out the difference between a teacher and an investigator; seldom are the power of imparting knowledge and investigation met with in the same man, and the first thing to consider in the case of a lecturer in our schools was that the man was a good teacher.

Yaws.

Mr. Jonathan Hutchinson, F.R.S., opened the discussion on Yaws, and showed a man from the West Coast of Africa believed to have been the subject of yaws. Mr. Hutchinson regards yaws as the parent of syphilis and adduced many arguments in favour of the contention. In Fiji, where yaws prevailed, there was no syphilis, and he argued that the presence of yaws prevented infection by syphilis. Mr. Hutchinson denies the statement that there is no primary sore in yaws. He believes that sailors and others becoming infected by yaws abroad came back to this country with the signs and symptoms of syphilis, the cause of the slight differences in symptoms being probably accounted for by climate.

Dr. Davies (Samoa) said that in Fiji children only were attacked by yaws, and he failed to see how the disease could be acquired by sailors. In Fiji and Samoa the natives considered yaws in children in the same light as we in Britain regard measles, that it was almost a certainty to have the disease and that the sooner it was over the better.

Dr. Manson considers Mr. Hutchinson's views on yaws open to question. He thought it strange that inoculation by yaws did not protect one from syphilis

and *vice versa*, if, as Mr. Hutchinson suggests, the primary poisons were identical. Why yaws was not seen in England was to be explained in the same way as the absence of many of the skin affections of tropical origin, viz., that a high constant temperature was necessary for their continuance and development.

Mr. D. C. Rees said that he saw syphilitic cases and yaws side by side in West Africa; that in the hinterland yaws was common and syphilis unknown, and that the natives in the West African Coast regarded syphilis as a new disease.

Colonel Macleod stated that he thought the case shown by Mr. Hutchinson was a case of syphilis, and all present agreed with him.

Dr. Phineas Abraham showed a case of leprosy in a young woman, and a girl suffering from generally diffused scrofulous lichen.

EXTRACTS FROM SANITARY REPORTS OF THE COLONY OF HONG-KONG FOR THE YEAR 1899.

By Dr. F. W. Clark.

Medical Officer of Health.

Beri-beri.

There were 197 deaths from beri-beri during the year, which number is somewhat in excess of the average for former years.

The increase occurred during the latter end of the year, and, although not amounting to an epidemic, gave cause for grave anxiety for a time. The following table shows the numbers of deaths that occurred among the Chinese during each month of the year:—

January	12	July	15
February	10	August	18
March..	11	September	16
April	11	October	24
May	11	November	29
June	15	December	25

The poorest quarters of the city were most affected with the disease, and deaths occurred in new as well as in old houses, and in houses fronting wide streets, as well as in those in narrow lanes; while several deaths occurred in matsheds put up for the temporary accommodation of workmen engaged in building operations.

A small outbreak of a disease which was considered to be beri-beri occurred, at the latter end of the year, in the Berlin Foundling Home. The following is a brief account of the outbreak.

It was stated that the Blind Home, a one-storey building, which contained about sixteen blind Chinese children, had had cases of beri-beri since July, and that the children from the Blind Home attended divine worship at the Berlin Foundling House. This latter house contained 102 children and girls up to the age of 16 or 17 years; and at the latter end of November two of the young children, both of whom were being surgically dressed—one for an affection of the eyes and the other for some skin affection—developed symptoms of beri-beri. Within a couple of days fifty to sixty other children were attacked with similar symptoms. On December 7 sixty-nine school-children, all of whom were suffering from this disease, with six big girls (who were in good health) to assist in looking after them, were sent to Macao, leaving twenty-seven healthy children in the house. Two of the children died in Macao shortly after their arrival there, but the remainder rapidly improved in health.

The main symptoms in these cases were dropsy and marked heart weakness, with in some cases a staggering gait and loss of reflex, but no marked evidences of paralysis, and it was suggested that the disease might be epidemic dropsy, but as many of the characteristic symptoms of this latter disease, notably the rash, the continued fever, and the evidences of intestinal irritation were also absent, it seemed more reasonable to suppose that the outbreak was one of beri-beri, especially as two or three of the patients, who were seen by various medical men in the Colony, were undoubtedly suffering from beri-beri.

The children who were attacked were all between the ages of 4 and 7 years, and all of them slept in a series of adjacent ground floor rooms. These rooms are thoroughly well lit and ventilated, and have close-boarded floors which are painted. Some children who slept on ground floor rooms in another part of the building were not attacked, nor were any of the girls who slept upstairs. No European cases of the disease occurred.

The children's dietary was a most generous one, comprising rice, eggs, fish (fresh and salt on alternate days), meat (beef or pork) at every evening meal, and thrice a week with the morning meal.

The special points about the outbreak seem to be the unusually early age of the patients (all between 4 and 7 years), the absence of overcrowding, the abundant lighting and ventilation of the premises, and the liberal dietary.

The twenty-seven healthy children left in the House continued in good health after the removal of the sick children to Macao.

Bubonic Fever.

The total number of cases of bubonic fever reported during the year was 1,486, and the total number of deaths was 1,428; this is equal to a case mortality of 96·1 per cent., as compared with a mortality of 88·2 per cent. in 1898, and 89·5 per cent. in 1896. I am inclined to think, however, that this increased mortality is more apparent than real, and that it was occasioned by the fact that a larger number of cases were able to escape from the Colony than in former years, owing to the temporary depletion of the Police service to meet the needs of the New Territory. The result of this was that only the moribund cases and the dead bodies were detected, while most of those who had any chance of recovery managed to make good their escape to Chinese territory. This view is borne out by the fact that while, during 1898, 36 per cent. of the cases reported were bodies found in the streets, &c., during 1899 40 per cent. were bodies so found.

The nationalities of the patients were as follows:—

Chinese	1,455
European (not including Portuguese) ..	7
Other Non-Chinese	24

Of the deaths from this disease two were British, one German, one Austrian, eight Indian, seven Portuguese, two Japanese, and the remainder 1,407 Chinese. The

mortality, therefore, amongst the non-Chinese alone was 67·7 per cent. as compared with 65·3 per cent. in 1898.

The same measures were adopted in dealing with the outbreak as had been used in former years, namely:—(1) the removal of the sick to hospital and of the dead to the public mortuary; (2) the detention of persons who had been in contact with the sick, pending the disinfection of the bedding and clothing; (3) the cleansing and disinfection of the infected premises, including a special house to house cleansing and disinfection throughout No. 9 Health District, in which the outbreak was most severe; and (4) the disinfection of all the public latrines by means of chloride of lime. The work was, however, greatly hampered by the inability of the police to render any assistance this year, as they had done in former epidemics, and the impossibility of obtaining reliable assistance from other quarters. In addition to the above measures an attempt was made to reduce the number of rats in the city by employing Chinese and furnishing them with traps and bait, but only some 1,000 rats were destroyed in this manner.

Enteric Fever.

Fifty-nine cases of enteric fever were reported during the year, as compared with fifty-two in 1898, but twenty-eight of these cases were imported, as compared with fourteen imported cases in the former year, so that there has been a slight reduction in the number of local cases of this disease.

The following table gives the number of cases reported annually since the introduction of compulsory notification:—

	Total.	Imported.	Contracted Locally.
1896	37	7	30
1897	65	23	42
1898	52	14	38
1899	59	28	31

Scarlet Fever.

Two European cases of scarlet fever occurred on board H.M.S. *Powerful* early in the year, the infection having apparently been brought out from home by some midshipmen who had recently recovered from this disease. The disease is comparatively unknown in this Colony, as it does not appear to occur among the Chinese.

Current Literature.

Notes on Tropical Dysentery.—This was the title of remarks by Dr. J. H. Musser, of Philadelphia, who related the case of a patient who had come from Porto Rico and was treated in the Philadelphia Hospital. There were marked emaciation and extensive pigmentation of the skin, with numerous boils upon the surface of the body. The symptoms were those of colitis and proctitis. The stools were free from fresh blood, and mucus was absent or but sparingly present. Purpura was a striking feature, with signs of scorbutus, which latter might have been the diagnosis made, were it not for the microscopical examinations. In agglutination methods the speaker thought we had a means of obtaining an accurate estimate of the affection.

Dr. Osler said he had formed the impression that the amœba would have to take a back seat since Dr. Flexner had reported his findings in connection with the bacillus he had described. He believed the disease was separate and distinct from that of the tropics, and thought it might be the same form as that which occurred in this country in epidemics seen in jails, hospitals, &c.

Dr. Briggs mentioned a patient who had come from Cuba, whose history was substantially that described by Dr. Musser for his own case, but here the Widal reaction occurred. In a second case it did not.

Dr. Hunter, of Minneapolis, spoke of cases from Manila, in one of which amœbæ were found. In a second case in which they were not found the symptoms were mild while the patient was at rest but severe under exertion.

Dr. Woodhull, of the regular army, recently stationed at Manila, spoke of the service to medicine at large, as well as to the army, rendered by Dr. Flexner and others who went to Manila to carry out these investigations. He found that many patients had to be sent to a colder climate before they began to show signs of improvement.

Dr. Murphy, of Missouri, said that in Kansas City he had studied three cases of amœbic dysentery. Expectorated pus showed that a liver abscess had perforated the lungs in one instance. He desired to call attention to the occurrence of the disease in northern latitudes.

Dr. Vaughan, of Ann Arbor, spoke of epidemic bloody flux occurring as far north as Michigan. Nothing more than the colon bacillus had been found in these cases, and he was inclined to think the latter the cause of this particular form of the affection. Strychnine hypodermically acted well in conditions of severe collapse and depression. Dr. Wilson, of Pennsylvania, referred to a case in which the tongue was denuded and appeared sandpapered throughout. The temperature was at times subnormal and never elevated. Emaciation was progressive till death, but there was never any pain. Treatment and dietetics were futile.

Dr. Flexner, in closing, gave credit to Dr. Woodhull for furthering the investigations in Manila by giving full opportunity to prosecute them. Every acute outbreak should be examined bacteriologically, and the blood should likewise be examined. The microorganism he described was closely allied to the typhoid bacillus, but it could be distinguished. Typhoid blood had no effect upon it. Liver complications in the form he studied were extremely rare.

News and Notes.

The Livingstone College.—After doing useful work for seven years, the authorities of Livingstone College find it necessary to obtain fresh premises.

At this institution men, not medical men, who intend to be foreign missionaries, are taught the elementary principles and practice of medicine and

surgery. They are not "medical missionaries" but the course is intended to teach missionaries how to care for their own and their fellow missionaries' health when far from a doctor, and how to deal with the more simple diseases of the natives of the country in which they happen to be located.

The College is appealing for some £6,000 whereby to acquire new and suitable premises at Leyton, in Essex. We are confident they will get the money readily, for the good work they are doing must recommend itself to every religious community.

FLEXNER ON THE BACTERIOLOGY OF ACUTE TROPICAL DYSENTERY.—On Thursday, Aug. 2, Professor Flexner, of Philadelphia, delivered an address on this subject before the Pathological section of the British Medical Association at Ipswich. Professor Flexner said it was erroneous to suppose that tropical dysentery was of a single kind. The disease had been attributed to different groups of organisms. The bacillus Coli communis was still believed by many to be the cause; the pyogenic cocci were regarded to be so by others, and the Amœbæ coli ranked as a potent pathological factor. Whilst investigating the diseases of the American soldiers in the Philippines he had succeeded in isolating a group of organisms (allied to the coli) not normally present in the intestines and showing peculiar reaction. When the acute form set up by these organisms became chronic the ulcers were superficial and were not undermined. Experiments showed that certain animals could be rendered immune with the products of these micro-organisms. Flexner believes the organisms he found are identical with those described by Japanese investigators as occurring in dysentery in that country. There seems some justification for the adoption of the term "infectious dysentery" as distinct from tropical dysentery.

WE are pleased to know that the Government are taking up the matter of the nature, causation and prevention of dysentery and enteric fever which have caused such havoc in the army in South Africa. An able staff of practical sanitarians, including Professor Trotter and Professor W. T. Simpson, are being sent out to join Major Bruce and to report upon the causes of the diseases due to misanitation in South Africa. A complete laboratory equipment is being sent out, and with scientists of the high standing in the profession which these gentlemen hold, there is every guarantee that the work will be well done. THE JOURNAL OF TROPICAL MEDICINE regrets the departure, for even a time, of one of the Editors, but the JOURNAL'S loss is the country's gain.

Letters, Communications, &c., have been received from:—

A.—Izett Anderson (Eastbourne).

B.—Dr. P. W. Basset (Haslar); Dr. Boddaert (Ghent); Dr. Osborne Browne (Gold Coast).

C.—Major R. H. Caster (London).

D.—Dr. R. Deduckson (Calcutta); Dr. A. B. Duprey (St. Vincent).

H.—Dr. J. Hewan (Edinburgh); Staff-Surg. V. G. Hooper, R.N. (China).

L.—Irving P. Lyon, M.D. (Buffalo, N.Y.).

M.—Dr. Preston Maxwell; Dr. Wm. Murray (Graaff Reinet).

R.—Dr. J. Ker Ramsey (Victoria); Dr. A. Raffray (Mauritius; Dr. Guthrie Rankin (London).

S.—Staff-Surg. Wm. Spry, R.N. (Mediterranean Station); Dr. Hy. Strachan (Lagos); Dr. Ella Scarlett (Korea).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

CONTENTS.—SEPTEMBER, 1900.

Original Communications.

THE USE OF QUININE IN MALARIOUS FEVERS.

By Rai Kailas Chunder Bose Bahadur, L.M.S., C.I.E.

Fellow of the University of Calcutta.

The subject has been almost exhausted, and any attempt to contribute further to its literature will, I fear, be simply an effort to bring into one place facts and arguments already urged by well known members of our profession. It is therefore necessary that one should be very brief and cautious in his remarks on the action of quinine in malarious fevers. In ancient times, when the properties of cinchona and its alkaloids were unknown to the practitioners of our country, they trusted chiefly to vegetable bitters in treating their fever cases. We now find that most of these bitters were taken from plants belonging to the natural order of Rubiaceæ or Cinchonaceæ.

To Dr. Twining is due the credit of first experimenting on the action of quinine upon the fevers of India. Its use has now become universally known, and the Kavirajes and Hakeems who were at one time dead against it employ it freely in all cases of fever, though under a different name. The late Kaviraj Roma Nath Sen used to say that the revival of the Hindu system of treatment and its progressive success in the cure of chronic malarious fevers dates from the introduction of quinine into the list of their febrifuges. The secret of success of the modern Kavirajes and Hakeems lies confined within the bottle of Howard & Sons' Quinine. It is now admitted by people and practitioners alike that the timely exhibition of quinine in malarious fevers can save lives from almost inevitable death.

In my practice I absolutely rely upon quinine in the treatment of malarious fevers, and the results, I am glad to be able to say, have always been most satisfactory. It is simply waste of time to wait for a remission or perfect intermission, for one cannot be too positive about the prognosis of a case of malarious fever, however mild it may appear at its onset. Instances are not unknown where apparently uncomplicated cases of intermittent fever have assumed a pernicious form and killed the patient. The use of quinine during the hot stage of fever has often been followed by hopeful results. In remittent fever it should be given in small repeated doses, for big doses are apt to cause sickness, insomnia, and depression of spirits. Failure of the heart's action, which is a serious complication of remittent fever, may be prevented by the judicious administration of quinine from a very early stage of the disease. The action of quinine upon the cardiac centre is really not depressant, but at the same time one cannot too confidently recommend it in cases where symptoms of heart failure have already set in, for the sickness and vomiting which sometimes follow the use of quinine tell seriously upon the function of this organ. I have not seen a single case where the administration of quinine in remittent fever has been followed by disastrous results. Intermittent fevers yield rapidly to quinine. One big dose of 15 grains shortly followed by another similar dose rarely fails to cut short the fever and prevent its return. I am not prepared to say whether quinine totally annihilates the plasmodia or simply makes them inactive and less capable of multiplication. The temporary absence of the parasites from the blood after a big dose of quinine is no

certain indication of their death. It is yet premature to say whether the plasmodia die out immediately on the exhibition of quinine or simply remain dormant for a time, and then under favourable circumstances revive and multiply. I would here cite an instance where a gentleman was subject to an annual return of intermittent fever (quotidian). The fever, as a rule, used to visit him during the month of June. It, however, yielded rapidly to quinine, and after a short confinement to bed for four or five days, he used to return to his duties. Under my advice he continued to take 8 grains of quinine daily for six consecutive months, and then went to Madhupur for a change. He returned to Calcutta in May and got the fever in June. He took quinine and got himself cured. The next year the fever returned in June and the patient obstinately refused to take quinine. The fever, which was intermittent before, soon assumed a remittent form of a severe type.

I have met with a few other instances of this nature. From such examples one can roughly conclude that the disposition of the malaria germs is very versatile and that they can, under different circumstances, assume diverse forms. The same variety which in one case produces *tertian* will in another produce *quotidian*, and from *quotidian* the fever may become *remittent*. It is not absolutely true that the large varieties will produce intermittent and the small ones remittent.

We have, through the observations of Laveran, Bignami, Golgi and others, been able to ascertain the cause of malarious fever and understand the character of its amœba, but we do not know what is the actual fate of these germs when the system is saturated with quinine. The symptoms and complications of fever depend mainly on the amount of poison they generate and not upon the special variety of the plasmodia. It is questionable whether one group of plasmodia produces one and only one special chain of symptoms.

There cannot be the slightest doubt that quinine is specific for malarious fevers, but at times its action upon an unmistakable case of intermittent fever is *nil*. I have very carefully observed that quinine in all its forms is inert in arresting or altering the character of double quotidian fever. Amongst numerous other cases, I give short notes of two where quinine failed.

Case 1.—Heeralall Batia, aged 32, a Hindoo Jain and dealer in Government opium, got fever with shivering at about 11 a.m., February 3. The fever subsided about midnight and the patient was absolutely free from it on the following morning. It returned again at noon and subsided during evening. The next day it came at 9 a.m. and left him at 3 p.m. It returned again during the night with shivering and left him with copious perspiration at 2 a.m. From February 6 to May 20, the fever followed one rule of coming twice within twenty-four hours, and then it altered its character and became very irregular in respect of the time of its coming and leaving. The temperature during the cold stage was 104° F, during the hot stage 104·5°, and at times it went up to 105·8°. The temperature of the night attacks was one degree less than of the day attacks. During the intermission it went down to 97°. Complications appeared in rapid succession and eventually the patient succumbed. Quinine was exhibited in large, moderate and small repeated doses, but to no effect. It was injected hypodermically, but the result was equally futile.

Case 2.—A respectable Hindoo lady, aged 16, married, living in a comfortable house, got fever, and placed herself under the treatment of an experienced officer of the Indian Medical Service and several other qualified practitioners. The fever was of double quotidian type; the diurnal paroxysms were preceded by distinct shiverings accompanied by retching and vomiting, but the night ones were absolutely free from these unpleasant symptoms. Her temperature ranged between 105° and 106° during fever, and it went down to 97° during intermission. She was treated with quinine and arsenic, but the result was extremely unsatisfactory. The spleen became hypertrophied and the patient soon became cachectic. She was sent up-country for change of air, but returned in a shattered state of health Quinine was given hypodermically for seven consecutive days and the result was disappointing. Cancrum oris and diarrhœa ultimately carried her off.

One special peculiarity observed in this case was that whenever quinine was given in fairly large doses the duration of the fever was prolonged and the period of intermission cut short. When given hypodermically it had a marked effect upon the temperature of her body, but none whatsoever on the duration of the fever. The poor patient suffered continually for eighteen months, and for the first twelve months the character of the fever was uniform. Afterwards it changed its type and became persistent. Since this alteration in the character of the fever the temperature did not rise higher than 103°. Epistaxis and bleeding from the gums preceded cancrum oris.

I have seen wonderful effects of quinine in pernicious intermittent fever which, when neglected, killed its victims in two days. Fortunately this form of fever is not usually seen in Calcutta. I have within the last twenty years of my private practice seen only ten cases. It has, like all other intermittent fevers, three distinct stages, but the intermission though complete does not last longer than three hours. The second paroxysm is graver than the first and the third is still more grave than the second, and the fourth brings on all the worst complications which, in the majority of cases, prove harbingers of dissolution. Quinine is the only thing to be relied upon in such cases. To discontinue quinine before the patient is rendered dumb, deaf and blind is not always a prudent course; for it is generally after the production of complete cinchonism that patients commence to manifest symptoms of recovery. The early exhibition of quinine is always advocated. Whenever we find that the prostration after the first paroxysm is very great and when the period of intermission is only nominal, we should at once take the case as serious and try to bring the patient under the influence of quinine. It is wise to err on the right side. To give quinine in small doses in such cases is to court disappointment. I would here beg to give reports of two cases where the neglect of giving quinine in the early stage of the fever was followed by disastrous consequences:—

Case 1.—A respectable Hindu lady, aged 35, had

fever with shivering. The cold stage lasted for an hour, and was then followed by the hot stage, which was very prolonged; temperature 106·4°. No pneumonia, bronchitis or pleurisy; urine normal; intense headache and delirium. These symptoms lasted for twenty-four hours, and then the temperature commenced to fall until it gradually went down to 98°. The period of intermission was short and lasted only for a couple of hours. The fever returned during the afternoon with its usual severity and lasted for thirty-six hours, followed by perfect, though short, intermission; 5 grs. of quinine were given at noon. At about 1 o'clock the patient complained of chill, soon followed by high fever. The temperature recorded at 10 p.m. was 107°. Along with the rise of temperature, head symptoms of a grave nature manifested themselves, and during morning, when a change for the better was expected, she had twitchings of the muscles of the face and rigidity of the forearms; her mind became confused and she died before evening.

Case 2.—Bany, a Hindoo gentleman, aged 28, had intermittent fever with short intermission and general prostration. Grave symptoms manifested themselves during the second paroxysm. Temperature went up to 108° and the patient became dull and apathetic; passed urine involuntarily; pulse 130—pretty good; respiration 26; no pneumonia. These alarming symptoms lasted for eighteen hours, and with the decline of temperature the patient gradually returned to consciousness. We commenced exhibiting quinine when the temperature was 102°, and in six hours the patient had 40 grains of muriate of quinine. We also injected 5 grains of the bisulphate hypodermically and steadily pushed on the muriate until the patient became absolutely deaf. The result was excellent, and he recovered, though after a protracted convalescence.

Quinine lessens the activity of the white corpuscles and gives tone to the red, and reduces the size of the spleen. Its success in the treatment of chronic malarial fever is illustrated by the two following cases:—

Case 3.—Sharbasia, a bigoted Jain, aged 50, suffered from chronic fever and hypertrophied spleen. He was absolutely anæmic with a hæmic murmur at the neck; gums spongy, susceptible to bleed on slight pressure; complete anorexia; vertigo and palpitation on slight exertion. Liver slightly enlarged; spleen painful on pressure and extending down to the umbilicus. Refused to take medicines other than stalks and leaves of indigenous plants. After repeated requests of friends and relatives he made up his mind to take quinine pure and simple. He was advised to try 5 grains of the muriate of quinine three times a day. The improvement was very slow at the beginning, but was very rapid after the expiration of a month. His appetite returned, and within six months from the commencement of the treatment he completely recovered his health. His spleen became normal; his anæmia and other unpleasant symptoms disappeared altogether.

Case 4.—Basantia, a respectable Hindu widow, aged 38, came under my treatment for enlarged spleen, fever and general weakness. She was anæmic; her menses scanty but regular; heart, lungs, liver, and kidneys normal. There was considerable wasting of the muscles; total anorexia; sickness and vertigo. She would only take quinine in honey and was accordingly permitted to take it in that fashion. Her improvement was most extraordinary, and when she returned to me after two months she was a new person altogether; her spleen nearly normal; no more fever, appetite fair, takes a lot of milk and digests it, can walk a mile without feeling fatigued or tired; sleeps well during the night and is comfortable in every respect. She had not a grain of iron in any shape and did not leave Calcutta for change of air.

To ensure the rapid and effective action of quinine, it is desirable that it should be given in the form of mixture; where 60 grains in pill form fail to cut short the fever, 20 grains in solution will succeed in producing the desired effect. The coated pills often take a long time to dissolve, and at times they are passed in their original form. As a rule children and delicate women strongly object to take quinine in liquid form. To induce them to do so it is necessary to cover its taste. I have seen powdered myrabolam when placed over the tongue and kept there for a minute entirely mask the bitterness of the alkaloid. Next to it comes pulv. glycyrrhizæ et zingiberis. When they are mixed up with quinine its taste is covered to a great extent. The former plan is good and harmless. In India myrabolam is readily procurable and is often used as a mild laxative by all classes of people.

The addition of salicine increases the potency of quinine and gives satisfactory results; its combination becomes useful when the fever is chronic, and when quinine alone fails to shorten its duration. I do not remember to have ever seen an instance where quinine and salicine failed to alter the character of malarious fever and check it materially. There is, however, one fundamental rule which should be carefully observed in administering quinine to a patient suffering from malarial fever, and that is to see the liver unloaded.

Case 5.—Monohor, a Hindu male, aged 32, resident of Rajpore, a village in the twenty-four Purgunas, came under my treatment for intermittent fever of six weeks' duration. The fever was of quotidian type; used to come regularly at 2 p.m. and leave at 11 p.m., with copious perspiration. Hepatic dulness slightly increased; spleen slightly enlarged; lungs, heart, healthy; no albumen in the urine. The patient stated that he gave a fair trial to quinine and got simply disgusted with it. He took it regularly for three weeks, but derived no benefit from it. I gave him 15 grains in one dose, and the fever returned as usual. The next day I combined salicine with it, 5 grains of each, and pushed this every three hours until he had taken 40 grains of quinine and 40 grains of salicine. The result was satisfactory. The patient had no unpleasant symptoms. The fever did not return. The following day he had no quinine, but 10 grains of salicine. The evening temperature recorded was 99°. Convalescence was rapid, and the patient soon recovered completely.

The presence of diarrhœa complicates matters, and quinine in any form simply aggravates the gastro-in-

testinal irritability and brings on complete anorexia. In such cases the exhibition of quinine should be withheld until the stools become normal.

Dosage of quinine.—The dose depends entirely on the character and duration of the fever. When the intermission is longer than twelve hours, small repeated doses agree well with the patient. When the period of intermission is short a full twenty-grain dose should be given at once followed by another ten-grain dose after a couple of hours. When alarming symptoms manifest themselves from the very early stage of the fever the patient should be cinchonised as quickly as possible. To wait for a perfect intermission is a mistake. Some complain of nausea or vomiting after a big dose, but there are so many things to check it that no special notice need be taken of it. To give quinine in big doses immediately before the paroxysm of fever is to court complications and troubles. The use of quinine through the rectum has not often been followed by satisfactory results. There is no doubt, however, that it is partially absorbed into the system.

Hypodermic injection instantly reduces temperature, but how this reduction takes place we cannot satisfactorily explain, for I have never seen perspiration follow this process; on the contrary, it is checked or entirely arrested by it. Deafness and noises in the ear, which sometimes become sources of annoyance and discomfort to patients, can be easily removed by the addition of ergot to the mixture. It does not in any way impair its action.

Quinine, when given to patients who are subject to hysteria or insanity, is liable to upset them. I have seen a young man, who some years ago suffered from mental aberration due to the excessive use of bhang, but who has since left off the habit, go mad after taking 40 grains of quinine in the course of twenty-four hours, but such cases are indeed very rare.

Dyspepsia and diarrhœa, which sometimes, though very rarely, come on after a prolonged use of quinine, disappear altogether after its discontinuance.

I have observed that quinine sometimes, though very rarely, causes diffuse dermitis, and the report of one or two cases will, I trust, be not out of place.

Case 6.—Gones Doss, a marwari, aged 55, came down from the Punjab to place himself under treatment for general dermitis said to have been of three weeks' duration. He said he took quinine for the cure of fever to which he was subject. He always took medicines from hakeems and never suffered from any unpleasant effects. This time, at the instance of a friend, he took quinine and got the disease. I never knew before that quinine could cause dermitis. I prescribed for him an alterative mixture which he continued for ten days, but found no improvement. I consulted my friend, Dr. K. MacLeod, then Professor of Surgery in the Calcutta Medical College, and prescribed for the patient an ointment containing resorcine, ferri sulph. and lanoline. The relief was almost immediate, and he was all right very soon and returned to his own place. Three years ago he came down to Calcutta on business and got fever. I prescribed for him quinine in 5-grain doses. After taking four doses he complained of an itching sensation all over the body, and on the following day he had a return of dermitis, which, however, yielded soon to resorcine and lanoline. On the appearance of the skin trouble quinine was discontinued, and the fever persisted for six weeks. During this time he took native medicines, but to no effect. His fever was quotidian. I gave him again one 15-grain dose in solution followed by another 10 grains six hours after the administration of the first dose. The fever subsided, but the dermitis returned and troubled him for weeks.

Case 7.—Mayiappa Chetty, another Hindoo Madrasee gentleman, of Canning Street, had fever and was treated with quinine; he took 40 grains in two days. He drew my attention to a sort of eruption of the nature of urticaria breaking out on his trunk and extremities. It had the same kind of itching as is commonly noticed in urticaria, but unlike it the rash never faded, but persistently remained for four days and was then followed by desquamation. He recovered; no ointment was used. On a subsequent occasion this gentleman had hemicrania, and I gave him 20 grains of quinine. There was a return of the skin trouble, which continued for three days, and then faded after partial desquamation.

MALIGNANT MALARIA, WITH URTICARIAL AND PETECHIAL ERUPTIONS.

By Charles Todd, M.D., D.P.H. (Cambr.).
Late Medical Officer, Mashonaland Railway.

Dr. Thayer, in his "Lectures on the Malarial Fevers," refers to an urticarial eruption of a peculiar morbilliform character, occurring occasionally during the paroxysm of malaria, but remarks that it should be remembered that this may not impossibly be due to quinine. He has also observed in several instances a slight petechial eruption.

These eruptions are rare—at any rate in cases met with in South Africa—and the following case seems worthy of record as the only one of over seven hundred cases of malaria seen by the author in Mashonaland, in which urticaria—apparently not due to quinine or other drugs—was observed.

The patient, a somewhat stout man of about 40, was first seen on the morning of February 4, in Umtali, Rhodesia. He had only been about six weeks in the country, but had lived some years in South America, and had never had malaria or any other serious illness. He was suffering from diarrhœa and vomiting, with some tenderness over the stomach, and was given bismuth, morphia and soda, but in the afternoon the pain had greatly increased, and when seen again he had severe colic with a temperature of 101°.

After a hypodermic of morphia the pain ceased, and next morning (February 5) his temperature was normal, and he expressed himself perfectly well, though somewhat exhausted by the vomiting and diarrhœa, ascribing his indisposition to some error of diet.

Next day (February 6), however, a vivid urticarial eruption appeared, covering the body, limbs and scalp.

This rash itched greatly, but beyond this inconvenience he complained of nothing except a slight tenderness over the stomach. The vomiting and diarrhœa had stopped, though the bowels were acting freely. The liver and spleen were not enlarged or tender. Temperature normal. Tongue thickly coated and breath foul.

During the day the rash behaved in a curious manner, fading in places and rapidly re-appearing in others and showing a peculiar multiform character. In many places it began as morbilliform patches, which rapidly extended outwards, at the same time fading in the centre and so producing annular patches of varying tints. In others, raised white patches appeared on a red base.

About ten o'clock that evening the temperature rose to 102°, and the patient became livid, with a running pulse and wandering delirium, refusing food and medicine.

He was freely stimulated with strychnine and digitalis, but early next morning (February 8) coma set in, with Cheyne Stokes' respiration and loss of corneal reflex. Temperature 98·6°. Several petechial spots also appeared on the chest.

Up to this time the case had been regarded as one of ptomaine poisoning, probably due to tinned food, as the patient had eaten some tinned rabbit the day before the illness began. It was, however, remarked that other persons who had partaken of the same dishes had not suffered. The question of possible malaria had been considered, but the fact that there was at the time no malaria in Umtali and that the attack did not begin with a rigor or any sensation of cold, and was unaccompanied by any swelling or tenderness of the spleen, rendered this improbable, especially as there had been very little rise of the temperature.

The successive crops of urticaria, however, coupled with the fact that the patient's condition was gradually becoming worse, suggested that the poison producing these symptoms was still being produced in the body, and the appearance of coma and the petechial eruption pointed to malaria as the cause.

An examination of the blood on the morning of February 8, showed the presence of malarial parasites in the form of small clear oval intra-corpuscular bodies about one-third of the diameter of the red blood corpuscles, each parasite containing two nucleolar-like granules. No extra-corpuscular parasites were seen.

Hypodermic injections of the acid hydrochloride of quinine were at once given, 39 grains being injected in the ensuing twenty-four hours.

In less than two hours the patient regained his conjunctival reflex and was able to swallow. He was fed on milk, eggs, brandy, &c., and began to sweat profusely, at the same time his temperature began to rise. In the evening the heart showed signs of giving out, the Cheyne-Stokes' respiration persisting and the temperature continuing to rise until early next morning (February 9), when death ensued from heart failure, the temperature reaching 104° immediately before death.

The continued low temperature—until after the administration of quinine—coupled with the petechial eruption resembling that seen in cases of malignant diphtheria, scarlatina and other exanthemata, and the heart-failure, all pointed to an intense poisoning by a toxin or similar body.

Whether or not the urticaria is to be attributed to the same cause is doubtful, but seems possible in view of its resemblance to the multiform erythemata produced by antitoxic serums.

Medical men practising in malarial countries are often accused of attributing every ailment to malaria, and in many instances they no doubt err on this side, but cases like the above emphasise the importance of bearing in mind the possibility of malaria, even in cases which at first sight do not suggest it; and the value of a microscopical examination of the blood in all doubtful cases.

In the above case it was subsequently found that the patient had visited the low country about a fortnight before his illness and he had presumably become infected with the parasite then.

ON THE ACTION OF LIGHT UPON THE HUMAN BODY IN RELATION TO DRESS.[1]

Communicated and translated by R. W. Felkin, M.D., London.

We think that all readers of the Journal of Tropical Medicine will be interested in a brief abstract of part of a paper (in so far as it deals with dress in the Tropics) which was read on April 27, 1900, by Jos Ritter von Schmaedel before the Anthropological Society of Munich.

After some remarks upon the action of light upon the bodies of animals and man, and sounding a note of warning that light is not a cure-all, as some would seem to think, he notes that the red end of the spectrum consists of heat waves, and that the violet and ultra violet, as the blue light waves, possess chemical action.

To go into detail, light waves between Frauenhofer's lines A-F on the spectrum are chiefly those which produce warmth. Those between F-H are chiefly those which produce chemical action. The light waves between F-H are obnoxious to many of those bacteria which convey disease to man.

Much may be said as to the direct or indirect action of light which is favourable to man, and light is necessary for man. But light, like everything else, has, so to speak, its shadowy side, and this side is too little noticed in practical life.

Von Schmaedel, basing his opinion on his wide photo-chemical experience, thinks that he is justified in stating "that the chemical action of the light waves exerts a solely beneficial action upon the living organism only when a certain balance obtains between them and the reaction which they induce.

"Just as it is possible for man to live within certain degrees of heat and cold, so the measure of the chemical action of light upon the balance of the functions of the organism is of the highest importance.

"If the amount of the chemical action of light is too great or too long continued a disturbance of balance sets in, which may at length, if too great, jeopardise the existence of the organism.

[1] See *Correspondenz Blatt der deutschen Gesellschaft für Anthropologie, Ethnologie und Urgeschichte. Juli*, 1900. No. 7.

"I think I may venture to state the hypothesis, that through long-continued chemical action of light one organism may be gradually overloaded with insoluble products of oxydation, which at length prevent their elimination and further depress the power of the serum to resist infection, causing changes in the formation of the blood, &c., &c. These are pure speculations, which have not yet been sufficiently investigated; their proof, however, is of so much importance that investigators would do well to pay attention to the subject." "In any case it is a matter of fact that the white man who proceeds to the Tropics suffers greatly from the intensity of the sunlight, and that he is not in a condition to live there continuously unharmed. Whether my view that this danger to health is intimately connected with the excessive action of the chemical light waves is correct I cannot say with absolute certainty, still I have grounds for this view based upon numerous observations on photo-chemical changes, and really think that such is the case. Man does not alone require light for his well-being, he must also guard himself from too much of it, unless he will run the risk of grave harm."

"The method by which we have to protect ourselves against an all too potent action of the chemical light waves, is placed at our hand by Nature in the most striking manner."

In 1887 the author read a paper on "Why are the Negroes Black?" in Munich, and pointed to the remarkable fact that "those races which inhabit zones in which the intensity of the light is exceedceedingly great, are all provided with a pigment in their skins which, in consequence of its colour, acts, one can only say, as an exceedingly effective protection to a too great penetration of the active chemical light waves."

"The black-brown pigment in the skin of the negroes, the brownish pigment in the skin of the Arabs, the yellowish and reddish pigment in the skin of other races—all belong in their various gradations to those parts of the spectrum which not only are not chemically active, but almost, if not quite, neutralise them."

"Nature acts exactly like the photographer when he seeks to protect his sensitive plates from the chemical action of the light. She surrounds the organism with a kind of dark room in order to paralyse too great an action of light."

Pigments whose colour are like those between Frauenhofer's lines F-H, neutralise the red or heat-producing light waves, *i.e.*, Frauenhofer's lines A-F, and *vice-versâ*. Pigments of a light white colour neutralise the heat rays, but let the chemically active rays pass unhindered; whereas black pigments permit the heat rays to pass, but stop the chemical.

"All these facts are of the greatest importance to individuals of the so-called white races who go for a long residence in the tropical or sub-tropical zones."

A white man who wears in the Tropics light white or blue clothes, has the advantage by their means of reflecting the heat rays, but he exposes himself to the full force of the chemical rays of light, which can pass completely through, and his health will be in a relatively short time highly endangered, as he has no sufficient protection in the pigments of his skin.

"That, not-to-be-despised, acclimatising fever which the Dutch call 'rothen Hund'—'the red dog'—consists in this, that the surface of the body everywhere is seemingly protected by light-coloured clothes, becomes exceedingly inflamed, causing severe symptoms of fever, is in my view a characteristic sign of the excessive morbid action upon the organism of the penetrating light waves."

Dark stuffs, so far as they do not fall under the "blue" side of the spectrum, or such whose colours fall between Frauenhofer's lines A-F, have, on the other hand, the advantage that they prevent the penetration of the chemically active waves of light, but they let pass unhindered the heat waves, whereby the well-being of the wearers naturally suffers in a high degree.

"It is therefore of importance—and according to my *lay* opinion of the greatest importance—for the civilisation of tropical countries, that the white races should construct a system of dress with the definite object of protecting the organism from the injurious influences, as mentioned above."

"In order to prove that white or coloured stuffs, which correspond to that part of the spectrum lying between Frauenhofer's lines F-H, permit the passage of the chemically active light waves in high degree, and that at the same time stuffs dyed black, or colours corresponding to that part of the spectrum lying between Frauenhofer's lines A-F, entirely, or at any rate partly prevent their passage—may I be permitted to show some examples of chloride of silver copies which have been made with these stuffs by means of a ten minutes' exposure to sunlight."

The speaker then showed a series of experiments which proved the above-mentioned theories — even four folds of white cloth did not prevent the penetration of the chemical rays to any appreciable extent, even after twenty minutes' exposure.

"You see, therefore, that the permeability of blue or white clothes is quite extraordinary, but that stuffs of the proper colour completely neutralise the chemically active waves.

"From this it follows that it is advisable for the white man who is compelled to live in the tropical zone, or one similar to it, to choose his clothes so that the outer surface is of a single plain colour or mixed colours or *broché* (material with a pattern) which would reflect the heat-light waves, whereas the inner surface should consist of a single or mixed or *broché* colour which would neutralise the chemically active light waves."

"This may be done by using either 'complex' material or material dyed different colours (reversible materials) on either side, or by the material being 'double-woven.'"[1]

The white man would also do well to base the colour of his tent, umbrella, &c., &c., upon the same principle.

By these means, even wearing the lightest clothing, an individual of a white race would place himself on

[1] *Note by translator.*—The same object may be obtained by wearing a "combination," or singlet and pants of one colour and coat and trousers of another. The only objection might be the heat the double garments might produce.

an even footing with the natives who are protected by the pigment in their skins from the strong injurious attacks of the tropical sun, and indeed without any danger to his organism.

Nothing more need be said as to the importance of this in the exploration, civilisation, and ruling of tropical countries.

In order to prove his point and to ensure correct colour being chosen, the author is patenting—*not* with a view of making money—a complete tropical outfit. He by this means wishes to investigate the matter as to whether the colours lying between A-F, &c., would be completely reflective, which colour or colours is best for the purpose, or whether only a percentage of the reflective waves of light would be either reflected or neutralised.

These are all small details, however, and von Schmaedel only wishes for a proof that his views are correct.

He invited discussion; but no report of that has as yet been published so far as is known.

ON PLAGUE AND ITS DISSEMINATION.

By Frank Tidswell, M.B., Ch.M.Syd., D.P.H.Camb.

An Address Delivered before the New South Wales Branch of the British Medical Association, April 27, 1900.

The Present Pandemic of Plague.

In the mountain valleys of Yunnan, in south-western China, there exists an endemic centre of plague. Our knowledge of it is of comparatively recent date, but it is to the effect that plague has been prevalent there since 1860, and probably from a date earlier still. When and how it first came there is quite unknown.

It is said as a rule there was little communication between Yunnan and the adjacent Chinese province of Kwang-si. In 1892, for military purposes, certain caravans left Yunnan whilst plague was raging, and upon reaching Long-Tcheou on the Canton River, certain of the muleteers from the Yunnan villages fell sick and died of plague. Infection of the town followed. In 1893 the disease recurred at Long-Tcheou; was conveyed down the Canton River to Naning-phu, and from thence overland to the seaport in communication with it, viz., Pakhoi. This route is said to be that of nearly all the traffic from Upper Kwang-si to Pakhoi. In February, 1894, plague appeared at Canton, and in April at Hong-Kong, to which places it may have been carried either along the Canton River, or by sea from Pakhoi. There was a recurrence at Hong-Kong in 1896.

Such is the history, as far as known, of the manner in which plague, after centuries of comparative quiescence, broke bounds so to speak, at the beginning of the pandemic in which we are now participating. From Canton and Hong-Kong the disease subsequently spread to other eastern places, *e.g.*, Macao, Amoy, up the China coast to the island of Formosa, the port of Inkou near Newchang, and to Moukden; to Port Arthur; to Kobe, in Japan; and quite recently to Manilla. There is said to be an endemic centre in Manchuria and Mongolia, North China, not far removed from Newchang and Moukden, but it is believed the disease came to these places not from Manchuria, but from Southern China.

The disease simultaneously extended itself in the other direction, and in 1896 appeared at Bombay. There is an endemic centre in India at Gurhwal and Damaon in the Himalayas, but this is not under suspicion as the source of the outbreak in India. The primary incidence of the plague at Bombay was at Mandvi, in a quarter near the Port, to which it is believed to have come over sea from Hong-Kong. From Bombay as a centre the disease spread overland to many towns and villages—Surat, Baroda, Ahmedabad, Palampur, Poona, Karad, Belgaum, Hubli, &c. Over sea it spread to Karachi, which also became a focus from which the disease extended into the surrounding country—Hyderabad, Kairpour, Rohri, Sukkur, Shikarpur, Jacobabad, &c. It is said, but disputed, that plague was present in Calcutta during 1896 and 1897, but this city was not officially declared infected till 1898.

Plague was reported at Bushire, Persia, in 1899. Although Kurdistan and Mesopotamia are notoriously prominent as endemic areas, the plague at Bushire is believed to have reached it from India, by way of the Persian Gulf.

Plague appeared in Russia, at the village of Anzob, near Samarcand, in 1898, and at Kolobooka, near Astrakan, in 1899.

In Arabia there is a noted endemic centre in the Assir district, Mecca and Jeddah having as evil a reputation for plague as for cholera. The disease was existent at Aden in February of the present year, probably imported over sea.

In Egypt cases of plague appeared amongst the crew of a vessel at Suez in 1898, and in 1899 (May) it broke out at Alexandria, but exactly how and from whence it reached this city is not known, opinion being divided between Jeddah and Bombay.

In the same year (1898) plague made its appearance at Tamatave, Madagascar; in Mauritius; Reunion; and at Lorenzo Marques on the East Coast of Africa. It is now existent at Cape Town. Quite recently Koch has asserted the existence of an endemic centre of plague in Uganda, Central Africa, which does not appear, however, to have played any part in the present pandemic.

Plague was prevalent at Oporto from June, 1899, to January, 1900. It broke out in the neighbourhood of the docks, and although its exact source is not known, it must, of course, have come from the East. It is believed to have been carried from Oporto to certain places in South America, having reached Santos and also Asuncion in 1899. It has appeared at Rosario and Buenos Ayres during the present year.

Near the end of last year plague became epidemic in Honolulu and New Caledonia. As you are aware, cases have been reported from Adelaide, whilst we in Sydney are not yet free from an epidemic.

From this brief, and necessarily imperfect, summary concerning the most recent extension of plague, it will be seen that the disease has once more assumed an expansive character. Even if we accept Hirsch's

suggestion that those of the sixth, fourteenth, and sixteenth centuries had their origin also in China (Cathay) the present distribution exceeds in geographical extent any previous pandemic with which we are acquainted. Plague has steadily made its way south of the equator, a fact which was observed with anxiety, and was acted upon by the Department of Public Health many months prior to the incidence of the disease on Sydney.

Its Mode of Dissemination.

"For the development of the disease and the formation of a plague centre there is always required the access of the specific virus of plague. Those plague centres extend just as far as the diffusion of the virus reaches to, but where the virus comes not, no matter how unfavourable the hygienic conditions, there the immunity from the pestilence is complete." In these views of Hirsch most of us would now concur, substituting the specific term *bacillus pestis bubonicæ* for the general word virus used by that author at a date long prior to the discovery of the micro-organism. The dissemination of plague, we would say, depends on the transportation of the plague microbe.

This microbe, as we have seen, is endowed with pathogenic properties, such as enable it to exhibit a very vigorous existence within the bodies of man and certain lower animals, more especially rats. Bacilli so contained could easily be carried over long distances by the movements of their hosts. But the diffusion of plague necessitates the passage of the micro-organisms from one host to another, and its comprehension demands consideration of the means by which such a transference can be effected.

The modes of exit of the bacillus from the bodies of the infected are said to be by way of the sputum (in pneumonic cases), with discharge from buboes, &c., with hæmorrhages, and with the bowel excretions and urine. It is not quite clear from the evidence to hand whether or not the bacillus is to be found in the excretions in the absence of hæmorrhage into the respective passages, and I am not acquainted with any data concerning the length of time the bacilli remain alive after such extrusion from the body.

The behaviour of the bacilli with regard to the establishment of a growth upon the artificial media of the bacteriologist has already been considered. With the exception of Hankin's statement that the bacilli lived seven months in a sealed tube I have no information as to how long growths will remain alive if untouched. My own cultures were too recently acquired to throw much light upon the point. By appropriate subcultivation the vitality of the bacillus can be maintained for long periods, but in this kind of saprophytic life the microbe is protected against the inimical agencies which would oppose its continued existence in nature.

With regard to the extra corporeal viability of the bacillus in other than laboratory media there is very little direct information, present views on this point being based for the most part upon evidence of an inferential character.

Assuming the bacilli to be shed in the ways indicated above, one would expect to find the bed or ordinary clothing, the room or the house, the soil, or perhaps water supply, implicated in the spread of the infection. As a matter of fact, linen and clothing have always been regarded with suspicion, and numerous instances of their apparent implication have been recorded, not only in olden days, but also during the present epidemic. For instance, in the case of a steamer from Bombay to London, and upon which two Portuguese were attacked sixteen and seventeen days respectively after arrival of the vessel in the Thames, the infection is supposed to have been carried in clothing which was unpacked only after reaching London. Collie mentions that a man having lost his wife from plague in Bombay took her clothing and jewels to a house in a village near Hurnai. In this house both his parents died of plague soon after, neither of them having been away from the village, and finally the man himself died. The English Commissioners consider it possible that the disease may remain latent in clothing, and vague mention is made by them of the detection by one observer of the bacilli on linen. I have not seen any mention of this elsewhere, and many observers deny the carriage of plague by such articles. It may be said with considerable certainty that those who handle the assumedly infected linen most, nurses and laundresses, are not specially liable to be attacked by plague.

With regard to the infection of rooms, no special observations have come to my knowledge. Hankin asserts that the bacillus will not survive long in goods on board ship, and presumably the same results apply to similar articles on land. He has shown, for instance, that if grain be infected with a pure culture, the bacilli soon die out; extracts were not infective after fifteen days.

Occasionally one meets with accounts which appear to indicate the persistence of infection in some parts of a house. For instance, there has been reported the death of twenty coolies from plague, caught whilst demolishing a house not previously disinfected. As such cases happen at places where plague is already prevalent, their exact significance must be open to question. Many authorities state that the infection is only present in the soil of the house, which in India is generally the floor. Yersin states that he found in the soil of an infected house, four or five centimetres below the surface, a bacillus which resembled pestis, but was not virulent. Upon one occasion Kitasato found dust from a plague house produce plague on inoculation, and Lawrie reports a similar experience. Presumably in these instances the occupants had recently suffered from plague. I have not seen any statement indicative of the length of time bacilli could be so detected after removal of their presumptive source. Kitasato asserted that the bacilli were easily killed by drying. It does not follow, of course, that either the soil or the dust of a house is dry, and moreover, Hankin states that the results of intratracheal injection have led him to believe that the resistance to dessication is by no means so slight as has been supposed. It is said also that plague may recur in the same house in successive years.

There is no doubt that plague may reappear in the same locality year after year, as witnessed by the recurrences in Hong-Kong, Bombay, &c., and the persistence of the disease in endemic areas. Plague "is

very often confined within a very narrow range, notwithstanding free and often busy communication with the neighbourhood." In the case reported by Francis, two Indian villages were situated not 500 yards apart, and apparently under exactly the same general conditions, but whilst one was attacked at every visitation of plague, the other always escaped. It is also noteworthy that persons who live upon the water often escape when the disease is raging close by on land. Such limitations of geographical distribution have been said to be hardly consistent with any hypothesis other than soil pollution. It may be questioned, however, whether the facts will bear this strict interpretation. Existence in the marshy banks of the Nile and in the arid desert of Arabia, in the warm soil of India and in the frozen ground of the Himalayas and Russia, &c., imply an adaptability to circumstances on the part of the bacillus which finds no support in the known facts of its life history. Moreover, plague epidemics exhibit a capricious incidence; places entirely escaping at one time may be severely afflicted at others. Again, plague disappears from most places after a period, not becoming endemic like typhoid. In view of these circumstances it must be confessed that, whilst there are certain features apparently incriminating the soil or dust of plague houses, there is no conclusive evidence to the effect that plague is a telluric disease.

Most authorities are agreed that the bacillus has a very brief existence in water. Hankin states that he once found the bacillus in a tank. As the result of experiments, Abel asserted the bacillus survived for several weeks. Wilm gave it twenty days in distilled, sixteen days in spring, and six days in sea water. The more recent observations of Gaffky showed that the bacillus did not remain infective in distilled water for more than three days, whilst in ordinary tap-water it was not discoverable after twenty-four hours. I have not seen any statements concerning multiplication of the bacillus in water.

The foregoing facts express the essential features of such information concerning the extracorporeal existence of bacillus pestis as I have been able to collect after careful search through all the literature at my disposal. It has to be confessed that with the possible exception of clothing and house-dust there is very little evidence that inanimate objects play any important part in the dissemination of plague. This view seems to receive confirmation from the statement that in the numerous experiments made by the German commission to test the saprophytic properties of bacillus pestis, it was found that in no case did its infective power survive more than seven days.

It is also supported by the fact that plague incidence is conditioned neither by season nor meteorological factors; at all events these agencies play no such obvious part as they do in certain other infectious diseases.

In addition to the facts already mentioned, opinion is practically unanimous that the bacillus readily succumbs to germicidal agencies. It is quickly killed by direct sunlight, and less rapidly by free exposure to air. The gaseous disinfectants act upon it quite as, or perhaps more, effectively than they do upon other disease germs, and it is easily killed by weak solutions of corrosive sublimate, carbolic acid, lysol, lime, mineral acids, &c. Nevertheless, in spite of the apparent ease with which the bacillus can be destroyed by very ordinary means, plague epidemics continue, no matter how vigorously such measures are applied. These facts suggest that the channels of plague dispersal are such as to protect the bacillus against the numerous influences which, if operative, would speedily ensure its extermination. Such security for the bacillus could scarcely exist elsewhere than in the body of its hosts, and one is led to think that the period of its saprophytic life, if it occur at all, is too brief and precarious to be at all significant from an epidemiological point of view. The transference from host to host must be more or less directly and rapidly accomplished.

As regards the human hosts of bacillus pestis, it has been abundantly shown that, save in the comparatively infrequent pneumonic form, plague is not conveyed by contagion. This is vouched for by various sets of facts; by the immunity enjoyed by doctors, nurses, and attendants upon the sick in hospitals, by the absence of cases amongst "contacts" segregated into camps or quarantine, by the absolute lack of association between successive cases, and by the fact that the disease is not specially incident upon overcrowded houses or areas, and does not for long remain confined to any particular quarter, but very soon begins to spread irregularly through a town or village. The suggestion afforded by the foregoing considerations is that there must be an intermediary host between man and man, and this *rôle* is now believed to be played by the rat.

As already stated, the rat is the one of all animals that most conspicuously shares with man the misfortune of being naturally susceptible to plague. In almost every place affected during the present pandemic, Hong-Kong, Formosa, Calcutta, Bombay, Karachi, Oporto, in South America, and here in Sydney, there has been reported great mortality amongst rats. In Hong-Kong, it is said, one man alone collected 20,000 rats dead of plague, and in Formosa the disease is known as "rat-pest." The association of rats with plague is by no means a modern observation, but has been mentioned by many of the older writers. Mr. Hankin tells us that one of the most ancient books of the Hindus (Bagavathi Purana) explicitly warns the people of the significance to be attached to mortality amongst rats. "On the moment rats fall down from the roof above (*i.e.*, out of their nests) and jump about and die, they (the people) will at once leave their houses with their friends and relations and will go to a plain." This direction is regularly observed by the inhabitants of Gurhwal, and also, it is said, by those of Yunnan, who, upon the occurrence of excessive mortality amongst rats, immediately vacate their villages and go into camps until such time as experience has taught them they may safely return. But the fact that rats sicken and die in large numbers during plague epidemics is, indeed, too well established to need further consideration in this place.

Most commonly, this feature constitutes one of the earliest indications of the presence of plague, and the primary incidence upon human beings occurs in the locality in which the rats are observed to be dying. It has been noted several times that the first persons

attacked were those working about wharves or in grain stores, places notoriously frequented by large numbers of rats. In Bombay the disease appeared in the quarter of Mandvi, and the victims were employed in grain depôts. In Oporto the first case occurred in a man who had been engaged in discharging a cargo of wheat. Here in Sydney, as you are aware, the earlier cases were all associated in some way or other with those wharves which receive most of the agricultural produce brought to the city. After a period of localisation to the vicinity of such places as those mentioned, the disease has usually spread slowly to other quarters, and this extension, in addition to the incidence on man, has usually been marked also by mortality amongst rats.

The other most significant epidemiological feature exhibited during this diffusion is that the principal incidence is upon the poorer members of the community. This partiality has been so obvious that plague has earned the title of *miseriæ morbus*. The locality and habitations and even the persons of the afflicted have been generally reported as indescribably filthy. Dilapidated houses, accumulations of refuse, and defective sanitary arrangements figure largely in all reports. They were existent in Canton, Hong-Kong, Bombay and Oporto, and of a surety Sydney cannot be cited as an exception. These conditions predispose to illness generally, but more pertinently to our present enquiry they attract and favour the entrance of rats into the houses. They are operative in the dissemination of plague only in so far as they do this, for plague does not necessarily attack the most insanitary or poverty-stricken parts of towns.

Apart from evidence of association of the kinds just mentioned, there are many instances which show that the presence of a plague rat was responsible for the illness in man. For example, a number of dead rats found one morning in a cotton factory at Bombay were removed by twenty coolies. Within the three following days about half of them fell sick with plague, whilst no one in the store not touching the rats was affected. Again, the coachman of an English family at Bombay found a dead rat in the stable and removed it. Three days later he fell sick with plague and died in a few hours. No other person in the same house was attacked. Various other observations of kindred kinds show only too clearly that the plague rat is a source of grave danger to human beings.

It is very commonly stated that whereas the rat is responsible for local dissemination, the disease is conveyed over long distances only by man. It is said that in many of the Indian villages outbreaks of plague occurred after immigration of a sick person or persons from infected towns. But it has been observed that such "imported cases" were often the only ones to occur, and where indigenous cases arose it was most commonly after an interval of weeks during which the rats began to die of plague. The same sequence of events has happened with regard to many seaports. To London and to San Francisco cases came in ships, but no outbreak followed, whilst at Capetown the arrival of a case on board ship seems to have been followed by rat infection and an epidemic. The transport of plague rats on ships is not merely a matter of conjecture, for according to Simond dead rats were found on the *Shanon* (Bombay to Aden) and *Patna* (Bombay to Karachi), and in each case were responsible for an outbreak of plague on board the vessel. There were also cases of plague on board the *Berenice* (Brazil to Trieste), *Polis-Mytilene* (Constantinople to Trieste), and *Taylor* (Brazil to New York), but in these cases nothing is reported concerning the rats.

Whilst not denying the possibility of some mode of extension from man to rats that is yet undiscovered, it is not necessary to assume that it need occur. Where man goes, rats also can, and generally do, go. Few if any ships are free from rats, and these animals are commonly carried about with merchandise of various kinds on land. The possibilities of such conveyance are too numerous and too frequent to be eliminated even by every practical effort that human forethought can suggest or human skill perform. It is very certain that as often as not there is no imported case in a human being, and the epidemic has, from its inception to its termination, expended itself entirely upon people who have never been away. The initial source of infection eludes the utmost vigilance, and that is precisely what rats would do. Infected rats getting ashore from ships would soon light up plague amongst their local associates, and thus lay the train for an epidemic.

Assuming the dependence of the importation and spread of plague by rats, there remains to be considered its decline. In most places the epidemic has lasted from three to seven months and then subsided, at times completely, but usually only to recur again some months later. This course it commonly pursues, irrespective of seasons or preventive measures, for as a writer has remarked, neither the elements nor the hand of man appear able to stay its progress. Observations upon rats again furnishes an explanation of the difficulty. It is well known that rats migrate often in large numbers, either in search of food or for some other object. It is also known that they will avoid for some time any place where a few of them have died from poison. It is asserted by several Indian authorities that rats similarly migrate from places where they are dying from plague. They carry the disease with them and so spread it, but continuing always to avoid the sick and dying, they at last become so dispersed that the disease dies out amongst them and only the healthy rats remain. Following out this instinct of self preservation, the rats of this particular generation are said not to return to their old haunts. Succeeding generations soon appear, and, slowly returning, re-occupy the evacuated quarters, which, as already mentioned, contain grain, refuse, &c., which attract them. If it should happen that the stragglers left behind at the time of migration have become free from infection all goes well, but if plague has persisted amongst them throughout, the incoming rats are again attacked, and then follows a recrudescence of plague in man. Simond asserts that the rats remaining behind become more or less immunised, and by retaining the infection in a mild form, thus keep it ready to break out when the progeny of the migrated rats return. In this way there is produced the succession of outbreaks so commonly observed in plague.

A great deal might be added to what has been said concerning the part played by rats in the incidence, dispersal, and decline of epidemic plague, but probably enough has been given to clearly show the nature of the evidence upon which the rat stands condemned as a source of infection for human beings. The validity of it has been amply conceded by Koch, Cantlie, Manson, Simond, Hankin, Simpson, as well as numerous other authorities, including many members of the Indian medical and civil services.

It is to be noted, however, that this contention has some opponents. It has been said that in many localities near Bombay the disease ran its course without a single rat being affected; and on the other hand there appears to have been an outbreak amongst rats at Kunkhal which ran its course and terminated without a single human being suffering. In the first case it may be that the incidence upon rats was merely unobserved, and the second obviously does not exonerate the rat from participation in other cases. It has been stated that comparisons have shown that the mortality from plague has been less in rat than in non-rat districts. Even if this be established, it will still remain to be demonstrated that in the latter places the part usually played by the rat was not assumed by some kindred animal. For, as already stated, Clemow has recently reported facts indicating that near Lake Baikal, in Siberia, a species of marmot (*Arctomys bobac*) has been instrumental in spreading plague. But, apart from debatable points such as these just referred to, it is beyond question that numbers of persons have got plague without handling a plague rat, and numbers of persons have handled a plague rat without getting plague. In any case, how does the bacillus pass from the rat to man?

Suggestions with regard to the site of entry of the bacillus comprise the digestive tract, the respiratory passages, and inoculation. As already stated, infection by way of the gastro-intestinal tract is a controverted question, and infection by way of the air passages is believed to occur only in pest-pneumonia, which never figures largely in epidemics. Consequently these two paths cannot be invested with any great epidemiological significance. Indeed, most authorities are in accord in regarding inoculation as the principal mode of infection. As you are aware, the interpretation placed upon the development of the bubo is that the inoculation occurs in the area from which lymph is collected to pass through the affected gland. The great frequency of femoral buboes is regarded as an indication that inoculation most commonly takes place through the skin of the lower extremity, and in China and India this selection was accounted for by the habit of the native population in going barefoot. They were supposed to become inoculated by infected dust through wounds and abrasions on the feet. But it has been found that in countries where such a custom does not obtain, the femoral region is still the most common site of the bubo, and consequently the theory of the "nu-pieds" no longer affords a satisfactory explanation. It is obvious, also, that none of the suggestions just referred to throw any light on the undoubted association of rats with epidemics of plague in human beings.

Upon this hitherto obscure feature of the dissemination of plague, we are now in possession of interesting data as the outcome of the researches of Simond. This observer noted that persons becoming plague-stricken after handling a rat did not necessarily develop their bubo in the axilla. As often as not in such cases the bubo was femoral. The same authority states that a plague rat is dangerous or not in accordance with the time that has elapsed since it died. If handled soon after death plague may follow, but if not touched for some hours, it may then be handled without risk. It was, says Simond, just as if the infection completely evaporated within a few hours after death. Finally, upon this point Simond observed that in many instances there was, on the area corresponding to the affected gland, a local lesion, a phlyctenule, which he regards as marking the actual site of inoculation, and in which plague bacilli are to be detected. The occurrence of such a phlyctenule is vouched for by several other investigators, though not all concur in Simond's interpretation of it. This local lesion is not always apparent, and, in fact, in the majority of cases it is not to be found; but this frequent absence, Simond contends, is due to the fact that it would only be produced when the inoculated bacilli were of comparatively mild virulence. Under such conditions there would ensue positive chemiotaxis, local leucocytosis and reaction, whereas if the bacilli were very virulent, the chemiotaxis would be negative, and no such local reaction occur. However this may be, Simond believes that the phlyctenule represents the point at which the bacillus found entry, and that it was produced by something that passed from the body of the rat to that of man, the said something being in his opinion most probably a flea.

He found by observation that perfectly healthy rats harbour very few fleas, and were very expert in removing them, but as they became sick they neglected their toilet, and fleas became more and more abundant upon them, so that they sometimes swarmed upon moribund rats. After death, on cessation of the circulation, and as the body becomes cold, the fleas leave it and seek another host. In this way he explained the "evaporation" of the risk attendant upon handling a dead plague rat. If the fleas from the dead rat reach another rat or a human being, they may inoculate the bacilli they acquired by ingesting the blood of their former host. In this way, according to Simond, the plague spreads from rat to rat and from rat to man. The man who handles a plague rat may be bitten by the flea, not on the hand or arm, but on the leg, and so the bubo be femoral. So it may happen that a person who has not had to do with a plague rat may yet be invaded and inoculated by the flea from it. It is enough that plague rats have recently died in the house or place.

By way of further substantiating his views, Simond demonstrated the presence of plague bacilli in the bodies of fleas from plague rats, and produced the disease by inoculating such fleas crushed up with sterilised water. To the still further objection, that the flea may contain bacilli in the stomach and yet not give plague by biting, Simond's researches supply the following answer. He placed in a large glass jar a sick rat, and also a healthy animal (rat or mouse), the latter being enclosed in a small cage so as to

prevent contact with the sick rat. If he left or placed fleas upon the sick rat, and allowed its body to remain lying in the jar for some hours after death, the healthy animal sometimes developed plague and died; but if he previously removed all the fleas from the rat and repeated the experiment, otherwise precisely as before, the healthy animal did not die, but remained perfectly well.

It must be admitted that Simond supports his flea theory at every possible point, and in doing so has performed a most interesting and suggestive piece of work. It still awaits confirmation by other observers, but meanwhile there is every reason to regard it as perfectly valid. Nowadays we accept the intervention of parasites with regard to many diseases, the mosquito in malaria, the tsetse fly in African horse-sickness, and the cattle tick in bovine tick-fever; and in none of these is the proof any more complete than that which Simond has furnished with regard to the flea and plague. The theory is perhaps not absolutely exclusive of other agencies. The bacilli have been found in bugs, flies and ants, but with the possible exception of the first, it is not likely that these animals play any significant part in plague epidemics. Admitting cases of accidental inoculation, of occasional contagion, of direct passage of fleas or bugs from man to man, and perhaps rarely the operation of some other obscure mode of conveyance, the great bulk of cases is not to be explained in any such way. Simond's hypothesis accounts for most of them and for much else that was formerly mysterious in the epidemiology of plague. The apparent infectivity of linen and clothing and of plague houses, and the influence of poverty, squalor and dirt, may easily be due to the harbourage such things afford for plague fleas. Even if we do not accept the evidence as entirely conclusive, nevertheless it is quite good enough to justify the adoption of the theory as a guiding principle in our efforts to combat plague. By so doing we lose nothing, and probably gain a great deal.

For if we agree with the dictum of Koch that plague is essentially and primarily a disease of rats, or as Manson puts it, that plague is a rat-borne disease, the direction which preventive measures should take is precisely that which will save us from the plague flea. I need not spend time in discussing such measures in detail. The destruction of rats and of all conditions which encourage or favour or facilitate their collection in the neighbourhood of habitations must be carried out by every means in our power. There must not be sudden and spasmodic onslaughts with intervals of quiescence, but a continuous, persistent, strenuous effort to suppress and keep suppressed the rat and everything that favours its existence. This course of action must be maintained after the cessation of an outbreak with a view of preventing its recurrence. No doubt the undertaking is a very large one, involving municipal and domestic cleanliness of a higher order than is usually considered necessary, but it is demanded of us, not only as a preventive of plague alone, but as the means of reducing the incidence of many other infectious diseases as well.

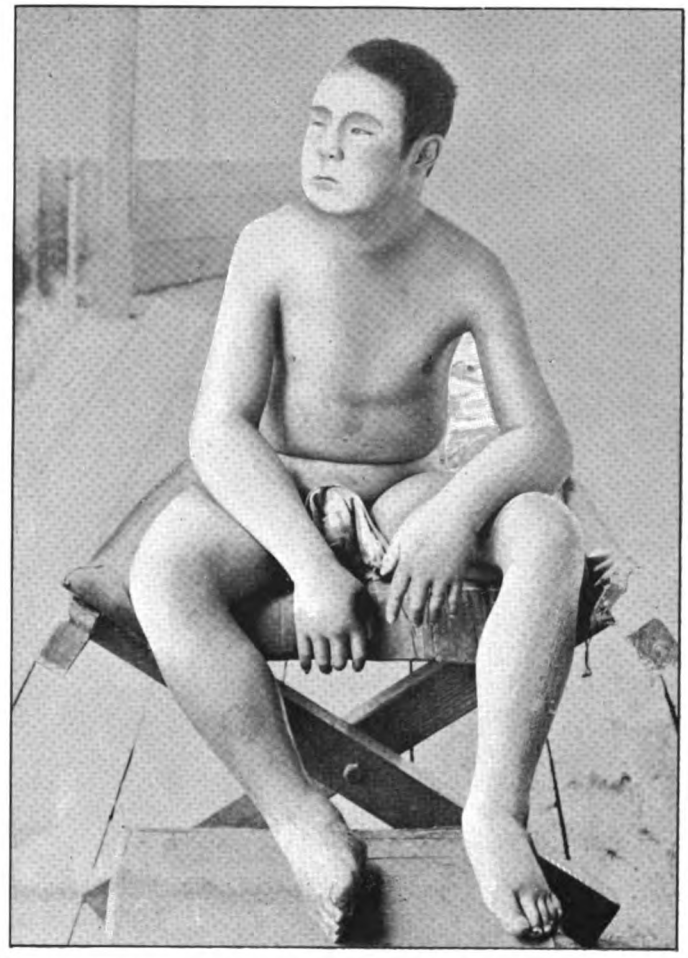
Upon the subject of prophylactics and curative serums I have nothing to say in this paper. You know what the prophylactic is, and most of you have had practical experience of what follows its injection. As to the protective value, I can at present add nothing to what has appeared in our journals. With regard to serums, the Department expects soon to test their efficacy, and I prefer to await the experience before expressing opinions on the subject.

You will notice that in compiling this paper I have not drawn much upon our Sydney experiences, the account of which will reach you later on through the proper channel. But I have no doubt you will have perceived, from facts within your own knowledge, that our epidemic parallels in most respects those recurring elsewhere, and that you fully appreciate what was said concerning fixity of type in an early part of this paper.

Notwithstanding that plague is an epidemic possessing such very clearly marked characteristics, it is a curious fact that there has nearly always been exhibited a tendency to deny its epidemic existence at its first appearance in any particular place. In the olden days people would assert that the disease was a "transmutation of malarial fever," or "typhus genius epidemicus," whilst more recently we hear it referred to as "malignant typhoid," or "continued fever with buboes," whatever that may mean, and in fact anything but plague. Dr. Neill Cook, the medical officer of health at Calcutta, remarks that the physician who diagnoses the first case is almost invariably ridiculed. The consequence of this attitude cannot be better expressed than in the words of a writer upon the outbreak at Alexandria. "The attitude taken up by the press is not to be admired. At one moment it denies that the disease is plague at all, jeering at the sanitary authorities for excessive zeal, and demanding a close time for the *chasse aux bubons;* at another moment, under the influence of hysterical excitement caused by the occurrence of a fresh case, it proclaims loudly that nothing is being done, and demands the burning down of infected houses, the absolute isolation of medical officers and all who have seen a plague case, the establishment of special commissions of intelligent citizens, &c., &c." I bring these matters before you without comment, and merely by way of showing that our own community has not been at all singular in its behaviour in the face of plague.

[We regret, that owing to a printer's error, the name of Professor Notter was mis-spelt in our last issue, in the notice referring to his departure for South Africa.]

SULPHUR AS A PROPHYLACTIC AGAINST MOSQUITO BITES.—In *Janus*, July-August, 1900, page 377, attention is directed in a note by Dr. L. Laloy to an observation that mosquitos refuse to bite a person who has taken sulphur. The experiment seems to have been carefully carried out, but as there are several reasons why mosquitos do not bite at times, it is impossible to come to a positive conclusion without further trial. We are aware of the antipathy the anopheles have to sulphur fumes. In January, 1900, we recorded the fact that in one locality of the Roman Campagna, where a sulphur spring perfumed the air with sulphurous acid gas for some distance around, no anopheles were found. The subject is deserving of further investigation.



BERI-BERI: ŒDEMATOUS VARIETY.
Japanese immigrant in Fiji.

BERI-BERI: ATROPHIC VARIETY.
Showing Muscular Atrophy and Drop-wrist. Japanese immigrant in Fiji.
Photographs sent by HENRY NOBLE JOYNT, Labosa, Fiji.

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THE

Journal of Tropical Medicine

SEPTEMBER, 1900.

PLAGUE IN GLASGOW.

THE appearance of plague in the city of Glasgow is of special interest in many ways. In the first place it is, so far as we know, the only outbreak of the disease which has occurred in Britain for over two hundred years. This in itself renders it an event of primary importance historically, and when we take into consideration the nature and locality of the outbreak the interest still further grows. We have had cases of plague brought to the London Docks on two occasions during the past twelve months, but in neither instance has the disease spread beyond the confines of the dock. A case of plague also occurred on board a ship bound from Cardiff to Hamburg quite recently. These board-ship cases, however, are on quite a different footing to those which have appeared in Glasgow. The locality in which plague appeared in Glasgow is practically in the heart of the city, away from the docks and wharves, and at some distance even from the Clyde. No direct communication between the shipping and the outbreak has been traced, for although the river is within 200 or 300 yards of the infected houses, the harbour ceases at Glasgow Bridge, which is situated lower down the river than the locality where plague appeared. It is therefore the absence of proof of direct contamination that renders the appearance of plague in Glasgow irrelevant and all the more alarming. It may be said plague is at present pandemic, and may appear anywhere; also that Glasgow is a large seaport, and it is naturally exposed to infection. These arguments, however, in view of what we know of the spread of plague, are insufficient to account for the disease occurring in a locality remote from apparent contagion. In searching for a cause, prostitution has to be thought of, but as the inmates of the abode in which the disease first developed were a child aged 3, the child's grandmother and grandfather, the proof of prostitution being the mode of infection is, to say the least of it, remote; and unless further light is thrown upon the source of infection the origin of the disease must be pronounced to be undetermined.

That the disease spread from a single focus of infection seems almost scientifically certain. All evidence points to a tenement in 71, Rose Street, on the south side of the river, as the initial centre; but how the bacillus pestis was imported thither is unknown. Two sudden deaths occurred in this abode during the first week of August, 1900. Wakes were held on the corpses, and it was amongst the visitors at this house, either during the illnesses of the patients or at the wakes, that the infection, which was subsequently diagnosed as plague, occurred.

Up to the present moment sixteen cases of plague have been admitted into the part of the Fever Hospital set aside for plague cases, and 113 persons have been isolated as "contacts." Since the disease was diagnosed one death only has occurred from plague, and further proof of

the power of "white" people to withstand the disease is thus furnished.

It is satisfactory to know that the disease was diagnosed early in the outbreak, that the sanitary staff and department is thoroughly competent, and that neither money nor pains are being spared to limit and eradicate the disease. The imposition of quarantine by a nation is a good test of its position in the intellectual and sanitary scale. The greater the ignorance of a people the longer and the more rigid is the period of quarantine they impose. This is being well brought out at the present moment, and a study of the rules and regulations employed against Glasgow vessels, affords not at all an unsound standard whereby to gauge the intelligent from the more backward nations of the world. It would appear as though the course of the disease had been stayed in Glasgow, but it is too early yet to come to a positive conclusion; and even if it does disappear for the moment a recrudescence is possible, and a careful watch will have to be kept for many months to come.

THE LONDON SCHOOL OF TROPICAL MEDICINE.

THE Autumn Session of this School will be opened by Sir William McGregor on October 3. It is an auspicious occasion in the history of the School, for it marks the commencement of the second year of its existence. Successful beyond all anticipation during the past twelve months, the future promises even better, and the decision arrived at that the School be enlarged is the best proof of the importance attached to education in tropical medicine, and the high estimation in which the instruction afforded by the School is held.

To Sir William McGregor especially, a school of Tropical Medicine must recommend itself. Although for a number of years Sir William has served his country as Governor in several colonies, he began his tropical experiences as a medical man. Like a few others of our profession, however, he laid aside medicine for administrative work, and the success which has attended his career in Fiji and New Guinea has amply justified his selection for the high office of Colonial Governor.

To a man with so wide and intimate an experience of the conditions of life in our Colonies, the advocacy of the claims upon the public of a school of Tropical Medicine will be a congenial task. He took up practice in Fiji at a time when the condition of our Colonies was considered of but little importance to the welfare of the mother country, and when their diseases were viewed as something almost apart from the science of medicine. Now all this is changed, and how completely changed may be gathered from an article which appeared in the *British Medical Journal* of September 8, 1900, in reference to the Plague Scare in London:

"It is perfectly certain that any case of plague eluding the vigilance of the Port sanitary authorities will be brought to the Seamen's Hospitals. The staff of the hospitals is not only on the alert, but the members of the staff consist of men who have experience of tropical diseases, and with the Tropical School at hand there is no danger of plague getting beyond the confines of the docks. When the London School of Tropical Medicine was instituted, there was a well-founded belief that it would prove of benefit to our tropical possessions; now it is brought home to us that the School is of direct benefit to ourselves, for there is but little doubt that if the hospital authorities had not had the advantage of its assistance these cases would have escaped diagnosis. With the hospitals at the docks so placed and equipped, and with a highly efficient Port sanitary authority, the public may rest assured that their welfare is being guarded, and that the enormous interests of the Port of London are in safe keeping."

SURGERY AT THE FRONT.

THE late Sir William Stokes, in a letter to the *British Medical Journal*, states that the Mauser bullet used by the Boers in South Africa is not the "harmless" or "humane" missile he was led to infer when first he landed in South Africa. He ascribes the change in the lethal powers of the bullet to be due to (1) the Boers converting it into a soft-nosed bullet by either slitting it longitudinally, or by removing a small portion of the apex; (2) the closer range at which the troops engaged during the latter part of the campaign, especially during fighting at night; and (3) that the physical endurance of the men towards the end of a campaign was inferior. Sir William Stokes also says that sepsis became more common as the campaign proceeded.

The results of the following operations show the possibilities of recovery nowadays compared with previous campaigns.

Case 1.—Compound comminuted fracture of the femur. Amputation at the hip joint. Recovery.

Case 2.—Compound comminuted fracture of both tibia and fibula; great laceration of soft tissues. Supracondyloid amputation of the thigh. Recovery.

Case 3.—Mauser bullet wound of the thigh, followed by the formation of a large aneurysmal varix in upper part of thigh; ligature of the common femoral above, and superficial femoral below, tumour in Hunter's canal; gangrene. Amputation at the hip joint. Recovery.

Case 4.—Extensive compound comminuted fracture of tibia and fibula. Teale's rectangular flap amputation of the leg. Recovery.

Case 5.—Severe bullet wounds of the shoulder, thigh

and leg; comminution of tibia and fibula. Amputation at the "place of election." Recovery.

Case 6.—Compound comminuted fracture of tibia and fibula; amputation at upper third of leg. Gangrene of stump; reamputation of the thigh by Teale's rectangular flap method. Recovery.

Case 7.—Compound comminuted fracture of the humerus. Modified circular amputation of the arm. Recovery.

Case 8.—Compound comminuted fracture of the humerus. Amputation at the shoulder joint. Recovery.

British Medical Association.

SECTION OF TROPICAL DISEASES.

Sixty-eighth Annual Meeting held at Ipswich, July 31, *August* 1, 2, *and* 3, 1900. *Colonel Kenneth Macleod, M.D. (Ret. I.M.S.), President.*

Papers read and discussions held on the first day of the Meeting, August 1, 1900:—

A DISCUSSION ON THE TREATMENT OF MALARIA BY QUININE.

I.—Andrew Duncan, M.D.Lond., M.R.C.P. (Major I.M.S. Retired), Physician to the Seamen's Hospital, Royal Albert Docks, London.

In opening the discussion on the treatment of malaria by quinine, I fear I cannot offer you many new facts as to its relation in this respect. I can but corroborate the facts already produced on its behalf. Owing, however, to the kindness of Dr. Manson—a name which I am sure you will agree with me is imperishably connected with the subject of malaria—I am enabled to adduce some fresh evidence in contradistinction to that previously in vogue as to its value in Africa.

The Prophylactic Action of Quinine.

In regard to its prophylactic action I may perhaps be permitted to recapitulate some of the evidence adduced in its behalf. Amongst French physicians M. Corré holds that the daily exhibition of quinine or tea given at the beginning of the malarious season, or whenever troops are operating in malarious countries, will diminish the chance of infection, but will not be of any avail against the more pernicious form. M. Marraud states that quinine gave good results during the Niger expedition, except as regards the gravest forms of the disease. MM. Kelich and Keur state that the officers of the French marines favour the exhibition of quinine, but again except the graver forms. M. Thorel had success in Mekong. M. Bizardel has found it successful in warding off attacks of pernicious ague. The Austrian and Russian surgeons, however, found no advantage in its use. M. Pola experimented on 736 soldiers, 500 of whom took quinine with attacks of malaria of 18 per cent.; the remainder not taking it were attacked in the proportion of 28 per cent.

As regards the American physician, Dr. Bryan found quinine and cinchona to be sure prophylactics.

Coming now to our own countrymen, Dr. Bryan was in its favour. My friend the late Surgeon-Major Parke, ten days before entering the mouth of the Congo, gave the officers 4 grs. daily. There were only two cases of slight intermittent fever, although 350 miles of the most unhealthy region were passed through. At Peshawar, in 1866, during the autumn, 1,203 men took quinine with an admission of 10·22 per cent. for malarial fever; 1,202 men who had no quinine had an admission rate of 27·28 per cent. for fever.

As regards my own results, they are gathered from the Sikhs and Goorkhas, so that no disturbing elements of race or station were involved. In 1889, whilst in charge of the 14th Sikhs at Peshawar, the regiment having suffered to an unusual extent from malarial fever during the preceding year at Thelun, A Company took from August 2 to October 31, 3 grs., increased to 5 grs. during the last week. B Company similarly took the same quantity of cinchona. C and D Companies took arsenic, of which more anon. The results were as follows:—

A Company,	taking quinine,	10	cases of	malarial fever
B ,,	taking cinchona,	11	,,	,,
E ,,	taking no drugs	24	,,	,,
F ,,		18	,,	,,
G ,,		21	,,	,,
H ,,		13	,,	,,

In 1896, 50 men of the 2nd Prince of Wales' Own Goorkha Rifles took 3 grs. of quinine, and had not a single case of fever. The men taking no drug had 6·5 per cent. of malaria.

In 1897, 50 men taking 3 grs. of quinine had no malaria. The men of the regiment who took no drug had 9·8 per cent. of malaria. In the Malay war no benefit was observed, or only a very slight one. As regards the West Regions of Africa, Harvey found no good. The bluejackets who took quinine had just as much fever as the men who did not. In the Ashanti wars of 1893 and 1896 it proved of no benefit.

Last year the results of an inquiry promoted by Mr. Chamberlain and Dr. Patrick Manson came to hand—133 answers were obtained, proving beyond a doubt that quinine does exert a prophylactic action here. Amongst the items that come out are the following:—

Of 44 persons who took it regularly 5 had no benefit and 37 had; of 16 persons who took it irregularly 1 had no benefit and 15 had; two recommend it for newcomers; two do not recommend it; two recommend it before the rains; one recommends it when feeling depressed; one preferred arsenic.

Of the whole number it was efficacious in 87·7 per cent.; there was no result in 12·3 per cent.

Comparison of Quinine Salts with other Drugs.

Arsenic.—The evidence of this is conflicting. In Italy Dr. Ricchi in 1883 gave it to 39 persons in the district of Borino, where malaria is very rife. Of these 36 escaped entirely, whilst the 3 remaining had very slight attacks. Of 39 people who did not take it the majority had severe attacks. In 1885 657 persons were experimented upon, 402 gave good results, 119 pretty good, whilst not much effect was obtained in 136.

Dr. Ralph Leslie, whilst acting as Government Medical Officer in the Congo Free State, administered it during fifteen days every six weeks. This rendered everyone immune who took it regularly.

I, however, have obtained in India no benefit. In 1886 half of the 23rd Pioneers from August to December took liq. arsenicalis with 28 cases of ague; the other half not taking it had 26 cases.

In 1887, from September 20 to November 16, the right wing of the 14th Sikhs took it and had 8 cases of ague; the left wing not taking it had 9.

In 1896 two companies of the 12th Sikhs took it from August 16 to October 30. They had 24 and 16 cases respectively; 4 companies not taking it had respectively, 24, 17, 21, and 13 cases.

Narcotine was recommended a few years ago at the meeting of the British Medical Association by the late Dr. Roberts. In 1896, 50 men of the 2nd Goorkhas took 2 grs. daily of the drug; they had 3 per cent. of malaria cases. In 1897, 50 men likewise had 6 per cent. The men taking quinine as just mentioned had no fever. So much for its prophylactic action.

Curative Action of Quinine.

For the last three years of my service in India I have notes of 867 cases. As regards the value of certain remedies,

my plan was to place each patient on a placebo for a week, and then, if the fever had not abated, to give an antimalarial drug. Considering only the cases in which the drug in question was given in attacks, 20 and upwards, quinine comes out best of all as regards the smallest time necessary for administration to stop the attacks, and as regards the number of failures.

In 78 cases quinine required on an average 2·11 days for the cessation of the fever, and had only 2·05 per cent. of failure.

Nim bark was given, either powdered in 1 drachm doses three times a day, or as 2 ounces of a decoction prepared by boiling 2 ounces of the bruised inner layer of the bark in a pint and a half of water for a quarter of an hour. This was the most successful after quinine. In 21 cases, the average duration of time in which the fever resisted treatment was 2·30, and its failures amounted to 18 per cent.

Berberis in 25 cases required 2·66 days for cure and 50 per cent. of failures.

Narcotine in 66 cases, in doses 2 to 3 grs., required 2·77 days for treatment, and had 1·06 per cent. of failures.

Kreat, a tincture of the kreat halviva, is much recommended by the Bombay physicians. In 42 cases it required 3·26 days for cure, and had 50 per cent. of failures.

Indergao was vaunted by a Bombay native physician for its "marvellous effect as a prophylactic of malaria." This may be so. As curative it stands lowest on the list. In 20 cases it required 4·6 days for cure, and had 5 per cent. of failures.

Method of Administration and Mode of Action of Quinine.

In most cases it was given by the mouth. In those cases, however, where after several days no result occurred, it was given by the rectum in 20-gr. doses as well. Here a cure in nearly all cases resulted. This plan of administration I can certify is most successful.

Quinine probably acts by destroying the organism. Dr. Richards, of Philipville, some years ago described this action. Some hold that its action chiefly is the small intracorpuscular forms and free spores, and so give it early; others, again, give it late, urging that it acts only on the large intracorpuscular forms. Another view is that it stimulates the phagocytes, the enemies of the parasite; but in whatever way it does act, it destroys the parasite.

II.—W. J. Buchanan, B.A., M.B., D.P.H., Major I.M.S. Superintendent Central Gaol, Bhagalpur, Bengal.

The prophylactic Issue of Quinine. A Synopsis of an Experiment on a large Scale in Indian Gaols.

The following is a statement of the case for and against the so-called "prophylactic issue" of quinine or other preparations of cinchona against malarial fever, as tried on a very large scale in the prisons of India during the past five years. In 1894 the experiment of giving daily, or several times a week, a dose of quinine or cinchonidine to every prisoner was tried in the Punjab. The sickness and mortality rates for the Punjab gaols in that year were so good and presented such a marked contrast to those of the other Provinces in that very exceptionally unhealthy and malarial year, that it seemed as if the good results must be to a great extent due to the issue of quinine. The Government of India therefore called the attention of the other Provinces to the system, with the result that a trial of the drug in this way was ordered in all gaols. The following account chiefly applies to Bengal, about which I happen to know most.

It so happened that the years 1895 and 1896 were healthy non-malarial years, even for the general population. The result was that at the end of the year a large number of most favourable reports were made on the use of the prophylactic. Most medical officers were, however, careful to point out that the general health of the public was in those years better than usual. My own experience of the prophylactic in the next year (1897) was in the Central Gaol at Buxar during a very malarious year, and I had no hesitation in stating that it had the effect of certainly reducing the severity of each fever case, though it did not prevent a large number of cases from coming to hospital. Also that the incidence of fever was much less than among an unprotected community in the neighbourhood of railway officials, and was decidedly less than in a village outside the gaol walls. Although in the gaol there were during the season about 500 cases of fever out of a daily average strength of 1,100, yet this proportion was, I was able to show, considerably exceeded by both the railway community and by the neighbouring village. Moreover, an examination of the nominal roll of those admitted showed that very few prisoners were admitted for more than once, and there were no deaths from fever or its sequela during the season. In 1898 I issued a series of questions on this subject to medical men in India, and got 51 replies. The details of this inquiry were given in the Journal of Tropical Medicine for March, 1899. Out of the 51 replies, no less than 47 gave their verdict in favour of the prophylactic issue of quinine. In the remaining cases the replies stated that the writers could see no appreciable difference between the health of prisoners in years when the prophylactic was issued and when it was not issued; but several answers specially noted that there certainly appeared to be a distinct reduction in the severity of the attacks. In 1898 the Director-General I.M.S., in his annual review of the state of health of the prisoners in the gaols of India, pointed out that the results could only be inconclusive so long as no control experiments were undertaken, and an editorial in the *Indian Medical Gazette*, September, 1899, strongly criticised the results up to date, and showed the necessity for regular control experiments, with the result that the following five papers, giving details of such experiments, were published in the *Indian Medical Gazette*.

These papers can only be very briefly summarised here.

(1) By Dr. R. S. Ashe, Superintendent of the District Gaol at Mymensingh. In this experiment both cinchonidine sulphate and Wrightia antidysenterica were used, for the double purpose of holding in check both fevers and bowel complaints. Dr. Ashe concluded from the figures obtained that there was "a diminished fever and dysentery prevalence," and this Dr. Ashe attributed to the use of these drugs.[1]

(2) By Captain C. J. Fearnside, I.M.S., Superintendent of the Central Gaol, Rajahmundri, Madras. This paper is specially important in that it gives for the first time an examination of the question from the point of view of the effect of the quinine on the malarial parasite in the blood. He points out from a series of blood examinations that quinine had no effect on crescents once formed, but that it had apparently an action on the prevention of crescent formation. He writes: "To conclude, quinine as a prophylactic is useful in those persons who have not been exposed to malaria, and, with one or two exceptions, I think the fever following the use of the drug is of a milder type and of shorter duration (due probably to an arrest of sporulation), and the readmissions of the same individuals for fever have been less numerous." It may be added that Rajahmundri had a reputation, before Captain Fearnside took charge, of being one of the most malarious gaols in India; so much so, that the numerous cases of malaria cachexia, with dropsy and anæmia, were even at one time considered as beri-beri.[2]

(3) By Lieutenant-Colonel Ffrench Mullen, I.M.S., then Superintendent of the Central Gaol, Rajshaye. He notes that in 1896 and 1897 he had reported strongly on the beneficial results of the issue of quinine, but that in 1898 he had to change his opinion, and that it appeared to him that the use of quinine had little or no prophylactic effect, and that it even seemed to increase the liability to bowel com-

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[1] *Ind. Med. Gaz.*, September, 1899.
[2] *Ibid.*, September, 1899.

plaints. It may be noted, however, that the gaol in 1898 certainly suffered from a widespread outbreak of influenza, and moreover there were no control experiments made.[1]

(4) By Major C. R. M. Green, F.R.C.S., I.M.S. This is a record of a control experiment made by Major Green, in Bankura Gaol, in 1896. A gang of 145 men were kept under the prophylactic treatment, and a gang of 180 were not under treatment; the other conditions were equal. The table quoted in the article shows that those under the treatment furnished fewer cases of fever than those not under treatment, and Major Green concluded that the prophylactic issue of quinine had "distinctly beneficial results."[2]

(5) By Major F. P. Maynard, I.M.S., then Superintendent of the Central Gaol at Hazaribagh. This paper gives the results of a very careful control experiment on a large scale. There were over 400 in each of the two gangs. Major Maynard gives tables to show the incidence of fever in the two gangs, and draws the following conclusions: (1) Cinchonidine does act as a prophylactic against fever when used in daily doses of 6 grs.; the frequency of the fever cases is reduced; more than one and a half times as many (7·72 : 4·88) cases of "fever" came to hospital from those not getting the drug, as from those getting it. (2) The severity of the cases is less; the proportion of cases admitted to hospital was nearly twice as great (5·82 : 2·85), and the proportion of admissions for remittent types was also nearly twice as great (58·8 : 30) among the prisoners not receiving cinchonidine, while their stay in hospital was decidedly longer. (3) Given in combination with dilute sulphuric acid and sulphate of iron the mixture has a useful effect in reducing the number of cases of diarrhœa and dysentery among the prisoners.[3] To these may be added the following figures, taken from the annual report of my own gaol (Bhagalpur Central) for the past year:—

A control experiment was carried out from July till November, 1899. In the cinchonidine gang there was a daily average strength of 1,050 prisoners and in the non-cinchonidine gang a daily average of 675. All cases coming to hospital for fever were recorded, with the unexpected result that an exactly equal proportion came from both gangs, namely, 222 cases of malarial fever, or 22 per cent. of the cinchonidine gang of 1,050, and 150 cases, or 22 per cent. of the non-cinchonidine gang. Still more strange, the results were also identical with regard to the numbers coming to hospital from both gangs for diarrhœa and dysentery. This showed that as far as these figures went no special benefit accrued to those taking the drug.

In conclusion, an examination of the above reports of an experiment on a very large scale is, on the whole, strongly in favour of the prophylactic issue of the drug as a preventive of malarial fevers. If Captain Fearnside's opinion that the prophylactic issue of this drug prevents the formation of crescents be further confirmed, an important argument in favour of such issue will be established. In the nature of things control experiments are more difficult to carry out over considerable periods than might at first be thought. Another point our Indian gaol experience has certainly settled, that is, that it is possible to daily administer preparations of quinine for many months at a time without the slightest mischief resulting. I have for the past five years been daily administering quinine or cinchonidine to, on the average, over 1,600 prisoners for the four months of the rainy season, and I have never met with a single bad result, even severe cases of quininism are conspicuous only by their rarity. It is needless to say that in spite of Professor Koch's alarmist views on the subject of quinine and hæmoglobinuria, there has not been a single case in my experience, nor have I, after enquiry, ever been able to even hear of such a case since the practice was introduced into the prisons of India.

[1] *Ind. Med. Gaz.*, October, 1899, p. 361.
[2] *Ibid.*, November, 1899, p. 401.
[3] *Ibid.*, March, 1900, p. 91.

III.—Robert Fielding-Ould, M.A., M.B., B.Ch. (Oxon). Liverpool.

The Administration of Quinine, with Special Reference to the Practice on the West Coast of Africa.

There is no subject, it seems, about which there is more difference of opinion in tropical countries than the value of quinine in malaria and the best mode of its administration. It was my lot last year to pass several months in the west coast of Africa, which has long been notorious as the most malarious district of the world. During my tour I had frequently the opportunity of hearing this subject discussed by medical men, but nowhere in the three colonies I visited did I meet with an even approximate consensus of opinion. That quinine is sometimes beneficial in malaria was generally freely admitted, but some were sceptical even on this point. As to the dose to be employed and the best modes of its administration, there was, I found, an extraordinary diversity of opinion. This section of the British Medical Association, by affording this opportunity for discussion of the various questions in their different aspects, is doing a good work, which will, I feel sure, prove of great benefit to practitioners generally, and indeed to all white men living in malarious countries.

The question of quinine administration is still an important one and has lost none of its importance in consequence of the recent discoveries which culminated in the conviction of mosquitos as agents in the propagation of the fevers. Fortunately, malaria is one of the few infectious diseases which, as is now universally conceded, possess a specific remedy; but its *modus operandi* has been the cause of debate ever since its first introduction into Europe by Del Cinchon. Since the discovery of the malaria parasites by Laveran, however, we have had many investigations made with the object of ascertaining the exact action of quinine on the hæmatozoa. Before this epoch-making discovery that malaria was due to a parasitic animal organism, it was known that quinine was very fatal to the infusoria, and indeed to all forms of lowly organised life; but even now we cannot go further than this, though in the case of the hæmatozoa I cannot help thinking that their death and subsequent disintegration are inseparably connected with the changes produced by quinine on the oxygenation processes normally going on in the circulation.

We know that these organisms low in the animal scale are greatly dependent on oxygen for the continuance of their life, and further, we know that one of the most important actions of quinine is to bind more closely to the hæmoglobin of the blood the oxygen present in it. The facility and readiness with which the oxygen is set free from the hæmoglobin is much diminished by the action of a dose of quinine. It is noteworthy, too, that quinine has a much more powerful influence on the hæmatozoa when they are young and in a phase during which they are chiefly concerned with their nutrition. This may be seen at any time by making continuous observations on the blood of a malaria patient after a full dose of quinine.

The unpigmented amœbulæ remain unpigmented, while those already containing pigment do not increase their pigment, showing that their nutritive processes are at a standstill.

A proof that the amœbulæ are incapable of development, though they may be morphologically unchanged, is afforded by the fact that blood rich in such parasites but strongly impregnated with quinine is incapable of reproducing malarial infection when injected into a healthy patient. This has been done by Bignami.

By many regular daily doses of quinine are administered as a prophylaxis against malaria. In India certain workers at the subject have made observations as to the value of this method and report in its favour. For my own part, however, I entirely disagree. It is obviously wrong to speak of the prophylactic effect of quinine, the alkaloids of cinchona do not *prevent* malaria, they only attack the

parasites when they are in a certain stage of their development.

A patient has not always young amœbulæ in his blood, yet he continues to take large doses of quinine, thereby often doing great damage to his digestion, the function of all others it is most difficult and most important to keep in good working order. Tropical climates are notoriously trying to the digestive organs, and certainly on the West Coast of Africa this is especially the case, where food is sometimes scarce and often most unsuitable; it is therefore highly undesirable that this habit of constantly taking quinine should be recommended.

Wordsworth Poole, of the West African Frontier Force, made certain experiments in Nigeria to test this method, and in his latest report says: "I am unable to satisfy myself that the advantages of quinine taken as a prophylactic outweighed the disadvantages of the dyspeptic symptoms produced by its prolonged use."

Nevertheless, there are many who hold a contrary opinion, and are able to take quinine with impunity. I have under observation a gentleman who has taken 5 grs. of sulphate of quinine daily for the last three years. He has had neither fever nor dyspeptic symptoms. I may add, however, that during the four years previously, when he did not take quinine, he was also free from both these troubles. He is an exception.

In addition to disturbing the peptic digestion, quinine is very apt to lead to congestion and torpidity of the liver, and it is precisely patients who exhibit hepatic symptoms who go down, when invaded by malaria parasites, with the pernicious forms of malaria, the bilious, remittent and hæmoglobinuric fever. Except in cases of actual fever, and on exceptional occasions (such as after great fatigue, or during a march) quinine should be avoided, and is useless as a rule, and often dangerous.

The time and method of administration of quinine are also questions which have led to much difference of opinion. Microscopical investigations give in ordinary cases the best indications as to time, and it may be laid down that in such a full dose of 15 grs. should be given when the parasites are about to sporulate, or are in their youngest phase as sporocytes. So long as the absence of vomiting or digestive disturbance permit it is best given by the mouth, but in other cases the hypodermic method must be employed. On the West Coast of Africa this method is not employed as much as one would have expected, though this is often due merely to the absence of the necessary syringe. Up to 8 grs. of the hydrochloride may be considered a full dose in this method, though very much more is often given with success. Some medical men in West Africa complain of the frequency with which suppuration follows this method of administration. I can only say that if proper aseptic precautions are taken this complication seldom occurs. In one case, however, of extreme cachexia, though every care was taken, suppuration followed. I am inclined to think this was due to the feeble condition of the patient. Of the intravenous method of Bacelli I have no experience, though it is difficult to see what advantages it possesses after these already mentioned. Lastly, I would draw attention to the great value of rectal administration. In West Africa this is not frequently employed, but in some cases it is most efficacious. After an enema 30 or 40 grs. may be injected with a little water or mucilage of starch, when it is rapidly absorbed, appearing in the blood in ten or twelve minutes. It is a pity, I think, further use is not made of this simple method.

In the treatment of malaria regard should be had, I think, for healthy persons in contact with the patient. So long as a malaria patient has parasites in his peripheral circulation, so long is he a standing menace to the health of the whole community. The administration, then, of quinine, should be continued for some weeks after the cessation of all febrile symptoms, and until repeated microscopic investigations show the blood to be free from parasites. In many cases, however, it is the custom to stop giving quinine too soon, leaving the gamotocytes free to be taken up by the mosquitos, and so propagate their species.

I make no mention of quinine hæmoglobinuria, of which many cases are on record. There is no doubt, however, that misapprehension and an imperfect knowledge of Koch's work has done much to revive in West Africa a dread of quinine, which had begun to disappear.

IV.—Patrick Manson, C.M.G., F.R.S., LL.D., M.D., Physician to the Seamen's Hospital Society, Greenwich.

Dr. Manson said: The reputed prophylactic action of quinine is but a phase of its therapeutic action; it is the application of the drug to the parasite, and not an immunising of the body against the entrance of the parasite we have to deal with; therefore we may confidently expect that if it will cure a malarial infection it will prevent its development—the development, though not the introduction of the germ. Further, that as some types of the parasite are highly amenable to the drug given therapeutically, similarly its prophylactic power will be greater against such; thus we may confidently expect it to be a timely prophylactic as against the benign tertian, but less active against the malignant tertian. The value of the drug is apt to be underrated in consequence of its being given in too routine a fashion, and often under conditions in which it cannot be absorbed, as in states of severe gastro-intestinal catarrh. Such failures should be eliminated in assessing its prophylactic value. I should recommend that in future experiments in prophylaxis be made with the aid of the microscope, and in reference to the particular type of malarial parasite it is used against, and also that the gastro-intestinal condition of the individual experimented upon be investigated and recorded.

V.—Lieut.-Colonel James Cort Marsden, I.M.S., Madras.

Lieutenant-Colonel Marsden said: I have invariably given quinine both as a prophylactic and as a therapeutic agent for the last twenty years, and the results have nearly always been satisfactory; but in reference to hæmoglobinuria, one case I had under my care some years ago made an impression on my mind. It was a private case, of which I have unfortunately lost the notes, and I speak from memory.

The patient was treated at Rajahmundry, the headquarters of a very malarious district; he was a German missionary who had been brought down from the Rumpa Hills. It was a case of malarial fever of the remittent type, with the usual symptoms. He was put on quinine as a matter of course, though much against his own wish, as he informed me that the drug had on a previous occasion produced bloody urine. I rather scouted the idea, but the urine shown to me at my next visit was undoubtedly hæmoglobinuric, blood being found by the usual clinical tests, and corpuscles apparent under the microscope. Each time the drug was repeated this symptom was observed; when the drug was omitted the urine was non-hæmoglobinuric. The case did not improve in spite of treatment, cold-packing, ice, &c., and the patient died from hyperpyrexia, with a temperature of 109° before death.

I may observe that this is the only case in all my service that I have ever observed such an effect produced by quinine. I myself still consider quinine our sheet anchor in malaria in all its manifestations.

VI.—David C. Rees, M.R.C.S., L.R.C.P., Superintendent of the London School of Tropical Medicine.

Mr. Rees said: Quinine was used as a prophylactic on a large scale in Nigeria by the West African Frontier Force in 1898. Unfortunately I have not the figures at hand, but as the quinine was not given under supervision on account of the force being divided into small detachments, the figures would not be very valuable. I, however, arrived at the following conclusion, namely, that 5 grs. of quinine administered daily, although that amount does not markedly reduce the number of attacks of fever, lessens their severity and

also the case mortality. I do not agree with Dr. Fielding-Ould's experience with regard to the sloughing caused by hypodermic injections of quinine; personally I have never produced sloughing by these injections, but the injection should be administered intra-muscularly not hypodermically, and with careful antiseptic precautions. When I proceed to a malarious country again I shall take 2 grs. of quinine three times a day, as I believe a small quantity of the drug circulating in the blood is more likely to act beneficially than one large single dose.

VII.—C. F. Harford-Battersby, B.A., M.D.Camb., Principal, Livingstone College, London.

Dr. Harford-Battersby stated that from personal experience he believed strongly in the prophylactic action of quinine. He and many others whom he had known had on early visits suffered severely from malaria, but on taking quinine prophylactically he had been free from fever, and others had been greatly benefitted. He quoted the case of Mrs. Bishop, the well-known traveller, who in travelling in malarial districts had stated that she took personally and gave to each of her servants a pill containing 1 gr. of quinine daily. None of them had ever suffered from malaria, even when they had travelled in company with other expeditions which had suffered severely. As regards the curative effects he urged the importance of giving quinine at the right time. He believed it to be the best course to see that the bowels were opened, and then to give 10 grs. of quinine at the commencement of the sweating stage, but in no case should the administration be delayed for more than four hours after the onset of the disease. He believed that the heroic doses formerly given were a mistake, and brought the medicine into discredit. With reference to possible evil effects of administering the drug, he alluded to the supposed tolerance of quinine, which was believed by some to result from its prophylactic use, and therefore to interfere with its curative use; this he considered to be without foundation. As to the suggestion that quinine produced hæmoglobinuria, whilst this might be possible, he did not believe that it could be regarded as a cause of hæmoglobinuric fever. The fact that quinine was given and had been given in large doses in all parts of the world, whilst West Africa appeared to be the home of hæmoglobinuric fever, which was seldom found in India, was a powerful argument in favour of this view. He regarded the view that quinine caused this fever as a most mischievous doctrine. He related a case in which quinine had cured severe vomiting in malaria, and believed that it could be found effectual in curing many symptoms which are malarial in origin although not so commonly associated with malaria. In conclusion, he dealt with the method of administration. He had employed Burroughs and Wellcome's tabloids with good results, and had never known them pass unchanged by the bowel. He considered it to be of some importance to disguise the taste of quinine, as the more nauseous methods of prescribing it might interfere with its toleration.

VIII.—Major E. M. Wilson, R.A.M.C., C.M.G., D.S.O.

Major Wilson said he did not think quinine had much prophylactic effect. In the Ashanti expedition of 1895-6 it was tried and discarded. In Sierra Leone, also, it did not seem to have much effect; in illustration of this he mentioned the case of a white non-commissioned officer, to whom quinine was administered by mistake by an orderly for fourteen days, and afterwards the man had fever. He had not found that the subcutaneous injection of quinine caused inflammation, and he had used it in many cases. No doubt Burroughs and Wellcome's tabloids were sometimes passed unaltered. No heroic doses were advisable; 10 grs. at a time were quite enough. He had never seen quinine cause blood to appear in the urine.

IX.—B. S. Ringer, M.D., Canton, China.

Dr. B. S. Ringer narrated a case of blindness due to quinine. A Spanish Roman Catholic priest, living amongst the Chinese up the country near Amoy, in the Tokien Province, had suffered from a severe and prolonged attack of malarial fever, for which he had taken large and frequent, but indefinite, doses of quinine. On arrival in Amoy he found him suffering from dimness of sight, and on the second visit he found him to be quite blind. The fever had, however, disappeared. Ten-grain doses of potassium iodide were then administered, and the sight gradually returned, and was eventually quite restored.

X.—Lieutenant-Colonel C. B. Maitland, I.M.S., Professor of Surgery, Madras Medical College; Senior Surgeon, Madras General Hospital.

Lieutenant-Colonel Maitland asked for information on the result of giving methylene blue in malarial fever. He gave it, in one epidemic, with even better results than from quinine. The drugs were given to alternate cases. No bad effects resulted from the blue, but the men's clothes and sheets became blue, and as the drug was procured with some trouble it was given up. The dose used was 3 grs. three times daily. In regard to the action of quinine on the pregnant uterus, and the teaching that the drug would either set up contraction or would keep it going when once it had begun naturally, his experience was that it could be safely given at any period of pregnancy, both in cases of ague complicating threatened abortion, and in cases of malaria in pregnant women suffering from another disease —for example, enteric fever. With reference to the giving of tabloids he had found that the hardest and most thickly-coated tabloid was effectual if broken first and given with a meal. When swallowed rapidly with water there was no taste.

XI.—James Cantlie, M.B., F.R.C.S., Surgeon, Seamen's Hospital, Royal Albert Docks, London.

Mr. Cantlie said we know so little concerning malaria in infants, and the action of quinine upon the suckling child when administered to the mother, that the following case may be instructive.

A child aged 3½ months, born of parents who had long resided in the South of China, but who at the time of the child's birth and afterwards resided in England, contracted a "feverish" attack which lasted six weeks, and defied all the usual drugs, change of residence, &c. The child was not teething; the mother was feeding the child wholly until well on in the illness, when cow's milk was given as well as the breast. A rise in temperature occurred every evening, followed by sweating during the night or early morning. As other plans of treatment had proved useless, the mother was put on 4 grs. of quinine thrice daily, and the child on ¼ gr. of quinine thrice daily. By the third day the child lost the fever and had no return. Examination of the mother's milk for the malarial parasite proved negative.

XII.—Edward Henderson, M.D., Shanghai, China.

Dr. Henderson observed that in the European population of Shanghai the benign forms of malarial poisoning are almost solely represented. No experiments in prophylaxis by the administration of quinine can be quoted as the cases are not sufficiently numerous. A dose of 15 grs. of quinine given in the sweating stage after the temperature had fallen, if followed by a few smaller (5-gr.) doses, is usually sufficient to put an end to an attack. With these small doses cinchonism is rarely troublesome. I have never seen any degree of permanent deafness nor any amblyopia follow the administration of quinine. Children suffer from cinchonism much as adults do if equivalent doses are given, but the effect may easily pass unnoticed from the child's inability to describe sensations. I think quinine decidedly a dangerous drug to give to pregnant women. In the old days I can recall two miscarriages which were produced, apparently, directly from large doses of quinine. Neither of the patients —they were both multipara—had ever had an accident of the kind before, and in neither case was any tendency shown

before the drug was given. I think the effect of quinine can be prevented, or at least lessened, by guarding it with some preparation of opium, or better still, chlorodyne. Chlorodyne possibly owes part of its value to the Indian hemp it contains, besides the morphine. The effect of Indian hemp in checking uterine hæmorrhage is, of course, well known. Hydrobromic acid or one of the bromides might be tried. If, as some believe, these drugs prevent tinnitus, supposed to be due to congestion of the labyrinth, they may conceivably exercise some influence over the circulation in the uterus. Personally I should not care to trust to them alone; they would need to be given in large doses.

XIII.—Major Ronald Ross, I.M.S. (ret.), Liverpool School of Tropical Medicine.

Major Ross pointed out that in old cases of malaria there might be a secondary form, due probably to enlargement of the liver and spleen —a form of a continued type not directly due to the parasites, and not amenable to quinine. This form had been noticed also by Vandyke. Carter, Kelsch, and Kiener and others, and was observed by the speaker while studying Kala-azar. Torti was the first to point out that quinine should be given before the access, and Major Ross cited an example in favour of the view with which he agreed. He considered that the drug should be continued for three months after infection, and that it was best given in solution.

XIV.—Guthrie Rankin, M.D., M.R.C.P., Physician to the Seamen's Hospital Society, Greenwich.

Dr. Rankin related a case where hæmoglobinuric fever was contracted, about fourteen months after settlement in Central Africa, by a young man who, during that time, took no prophylactic.

The patient was invalided home, and experienced a mild attack after his return to England. He returned to Africa after four months perfectly well, and during the following two and a half years he suffered no return of malarial trouble. During the whole of his second residence he took quinine daily, in saltspoonful doses. He is now at home in excellent health, but has suffered for some months from a persistent dermatitis of both hands, which he ascribes to the long use of quinine.

Dr. Rankin wished to inquire whether, in the experience of those who had largely used quinine in the tropics, the widely expressed experience of the absence of cinchonism could be accounted for by the fact of its administration to patients suffering from malaria, because in the practice of every physician at home cinchonism, coming after comparatively small doses of the drug, was not uncommon.

XV.—Oswald Baker, M.D. (Lieutenant-Colonel I.M.S., ret.), Physician Seamen's Hospital, Albert Dock, London.

Dr. Baker stated that quinine was given in this country in conditions other than malarial fever in doses aggregating 15 grs. daily, extending over long periods of time, without any prejudicial effect whatever. He thought the reason quinine so often failed as a prophylactic was because it was not administered in sufficient quantities. He thought it would be desirable to elicit the opinion of this meeting on the subject. He was of opinion the prophylactic dose was the same as the curative dose.

XVI.—Colonel Kenneth Macleod, LL.D., M.A., M.D., Professor of Clinical and Military Medicine, Army Medical School, Netley.

Colonel Macleod remarked that Dr. Manson had pointed out the importance of using the microscope during the administration of quinine as a guide and check. This was, when practicable, obviously desirable. But the practice involved considerable labour, and in cases in which the parasite was not present in the peripheral circulation might be accompanied with doubt and disappointment. In these cases, as Dr. Manson now reminds us, the pigment and pigmented leucocytes might be seen. Koch recommended another and more extended use of the microscope as a means of detecting malarious infection in a community. This system of exhaustive examination of the blood of a community, he contended was necessary, because parasites might exist without any other pyrexia or other overt indication of malarial infection. This mode of using the microscope was still more laborious, and can only be possible under special circumstances in small communities; but if the extirpation of malaria by means of quinine was attempted it must be resorted to. The use of the microscope in this matter, however important, had its restriction. Dr. Battersby had drawn a very important distinction between hæmoglobinuria and hæmoglobinuric fever. We knew that hæmoglobinuria was caused by the adminstration of many poisons, inorganic and organic, and it was quite possible that quinine might, under certain circumstances and in certain persons, give rise to hæmoglobinuria, but that was a very different matter from quinine giving rise to hæmoglobinuric fever, which is a pyrexia of very definite type, of which the presence of hæmoglobin in the urine was but one of several very characteristic features.

ON THE METAMORPHOSIS OF THE FILARIA SANGUINIS HOMINIS IN MOSQUITOS.

Especially with Reference to its Metamorphosis in the Anopheles Rossii and other Mosquitos of the Anopheles Genus.

By S. P. James, M.B.Lond.,
Captain I.M.S., Quilon, Travancore, India.

Dr. Patrick Manson's discovery of the metamorphosis which the filaria Sanguinis hominis (the embryo of the Filaria Bancrofti) undergoes in the bodies of certain mosquitos, has recently been confirmed by Dr. Bancroft in Australia; and I desire in this paper first to make some remarks on the time necessary for this metamorphosis in different mosquitos, and afterwards to describe briefly the experiments which I have carried out on feeding mosquitos on the blood of filariated persons, which prove that mosquitos of the Anopheles genus are capable of acting as efficient intermediary hosts for this parasite.

Dr. Bancroft discovered that mosquitos can be kept alive in confinement for many days by feeding them on bananas; and having thus overcome the chief difficulty which has, up till now, prevented the confirmation of Manson's discovery, he was able to prove that in a certain species of the Culex genus of mosquito (Culex ciliaris, Linn.), the metamorphosis of the filaria required seventeen to twenty days for its accomplishment.

The important difference between his results and those of Dr. Manson is, that whereas Manson stated that only seven days are required for the metamorphosis of the filaria in the mosquito, Bancroft found that in his mosquitos more than double that time was necessary, and he comes to the conclusion, therefore, that Manson must have been in error in his estimate of seven days, and is of opinion that Manson's mosquitos must have fed on the blood of a filariated man some weeks before he began his experiments with them. I do not think that this conclusion is justifiable. If it is correct, it means that Manson employed any mosquitos that came by chance into his filariated patients' room, and was not careful to ensure that they had not fed on an animal or on man before they entered the room.

But Manson wrote distinctly as follows:—

In experimenting with the mosquito there are two points I was careful in attending to; I employed the proper species of insect, and I took care that its only food was the blood of a filariated man. Those who would repeat my experiments must bear these two points in mind.[1]

Thinking as I do that Manson would not have said this

[1] *Transactions of the Linnean Society of London*, Series II., vol. ii.

unless he was sure that the mosquitos he employed had not fed at all previous to their meal of filariated blood from his own patient, and because I have myself found that in the Anopheles Rossii and in another species of the Anopheles genus of mosquito, only twelve to fourteen days are required for the same metamorphosis, I do not believe it impossible that there may be, in a place where filariasis abounds, a species of mosquito in which even in seven days the metamorphosis can be effected. It will be seen later in this paper, and it has been shown by other observers, that several different kinds of mosquitos are probably efficient hosts for the filaria nocturna, but it is probable that those kinds are not all equally efficient—in some the metamorphosis would take longer than in others.

The most efficient kind of mosquito would very probably be found in a place where filariasis is exceedingly prevalent, for it is reasonable to suppose that the capacity for acting as an intermediary host for this parasite would increase, from generation to generation, in a favourable species of mosquito, in a place where these mosquitos were always acting as intermediary hosts.

Thus it is likely that in the place where Dr. Manson carried out his experiments a very favourable—perhaps the most favourable—species of mosquito would be found, for filariasis is exceedingly common there. One does not know how common the parasite is in the place where Dr. Bancroft's experiments were made; but, from his statement that he only knew of one person in whose blood filaria embryos were present, one judges that it cannot be very prevalent there.

It would appear probable that if the "common house mosquito" of Australia were the usual intermediary host of the filaria nocturna, filariasis would be more prevalent there than it apparently is. One must remember, too, that climate, and probably other factors, have an important influence on the time taken by parasites to develop in their intermediary hosts; and it is possible that, as some of Dr. Manson's mosquitos were kept at a uniformly high temperature (80° to 85° F.) in an incubator, they were more favourably placed, in respect to climate, at any rate, than Dr. Bancroft's, for the latter states that when the weather was cold as long a time as thirty-five days was required for the metamorphosis to be effected in some of his mosquitos.

For the above reasons I think that the time required for the metamorphosis of the filaria nocturna in the mosquito cannot be stated definitely, and I consider that the "filaria-bearing" mosquito of Dr. Manson was probably a more favourable species than the Culex ciliaris of Dr. Bancroft.

The following is an account of my experiments on feeding mosquitos in filariated blood. All the experiments with Culex mosquitos, and many of those with Anopheles mosquitos, were carried out before I was aware that Dr. Bancroft was also working at this subject.

The mosquito which Manson found to be an efficient intermediary host for the filaria nocturna belonged to the Culex genus, and for this reason I commenced my experiments last year on mosquitos of this genus. At that time I employed the following method:—

A number of female Culex mosquitos bred from larvæ were placed at night under the mosquito curtains of the bed of a man in whose blood filaria embryos abounded. In the morning these mosquitos which had fed were transferred to large glass vessels in which bananas were hung. These bottles were kept in the open air in a shady place, and each day the mosquitos which died were removed and examined in the manner recommended by Dr. Manson. Thus from day to day I watched the progress of development of the filariæ. Probably owing to my not renewing the bananas sufficiently frequently, none of the mosquitos used in these experiments lived more than twelve days, and out of over 200 mosquitos that I fed in batches on different nights for this series of experiments, only 12 or 13 lived that length of time. Among these, however, there were specimens of two species in which the filariæ had reached an advanced stage of development. They measured from $\frac{1}{80}$ to $\frac{1}{40}$ of an inch in length; the alimentary canal was very distinct, and the head and anterior part of the bodies exhibited lively side to side movements, together with quivering movements of the whole body and constant flicking movements of the tail. Intermediate forms of the filariæ between these and the initial stages were found in abundance in other mosquitos of these two species, which had died or been killed on earlier dates. I think it probable, therefore, that had these mosquitos lived some days longer, the final stage of the parasite would have been reached in them, and they would have proved to be capable of acting as efficient intermediary hosts of the filaria.

Specimens of these mosquitos were sent to the British Museum and to Major Giles, I.M.S., for identification, and have been pronounced to be (1) Culex microannulatus; (2) Culex alleopictus (Skuse).

These experiments, though satisfactory as far as they went, could not be said to have been quite successful, for the mosquitos died before the final stage of the filariæ was reached, but they proved that in two species of Culex mosquitos the filaria nocturna can live and develop to an advanced stage.

I was influenced, however, by Manson's statement that seven days only were required for the metamorphosis in the proper species of insect, and having noted that in the district of Shertully (where over 40 per cent. of the inhabitants are "filariated"[1]) mosquitos of the Anopheles genus are very prevalent in houses, I now commenced feeding Anopheles mosquitos on filariated blood.

I found, however, that it was even more difficult to keep these mosquitos alive in confinement than Culex mosquitos; they do not readily feed on bananas, and it was not permissible to keep them alive by feeding them on animals or birds, lest some other parasite might be introduced as well as the particular filaria one wished to study.

After many trials, I adopted the following plan, which also has the advantage that different stages of the parasite are present in the mosquito at the same time, and the connection between the stages is clearly seen.

A number of female Anopheles mosquitos bred from larvæ were placed at night under the mosquito curtains of the filariated man's bed. In the morning all that were full of blood were transferred to bottles in which ripe bananas were hung. In the evening they were replaced inside the curtains with the filariated man, and the next morning collected again into the bottles. This practice was continued for the first five days and nights, so that each mosquito had at least four or five meals of filariated blood. After the first five days the mosquitos were taken to my bungalow, where I fed them for the first three or four nights by liberating them under the mosquito curtains of my own bed, so that they obtained meals of ordinary blood instead of filariated blood. In the daytime they were kept in the bottles with bananas. After the ninth or tenth day they obtained no more meals of blood, but were kept in the bottles with bananas until they died.

In this method a good many die or are lost in the curtains, but as when fed on bananas alone none of my Anopheles mosquitos lived more than five or six days, it is, I think, the only way by which they can be kept alive for any length of time. It was possible in this way to keep them alive for at least fourteen or fifteen days, and some lived to the seventeenth and eighteenth days. Each day the mosquitos that died were examined by breaking up the thorax and abdomen on separate slides with needles in a weak saline solution, and examining with a $\frac{1}{3}$ inch and $\frac{1}{6}$ inch objective without applying a cover-glass.

By this method I have been able to trace the metamorphosis of the filaria to its final stage in the Anopheles Rossii, and in one other species of the Anopheles genus of mosquito, the name of which I have not yet ascertained.

[1] See article by the author in *Indian Medical Gazette* of 1900.

Before I had read Dr. Bancroft's paper suggesting that the filariæ may be introduced into man while the mosquito bearing them is in the act of biting, I constantly omitted (as I think previous observers had also done) to examine carefully the head and proboscis of my mosquitos. Lately, however, I have been careful to do this, and the result shows that an examination of these parts is, from the point of view of the method of infection, exceedingly important.

To attain the final actively-moving stage of the metamorphosis, requires in the Anopheles Rossii from twelve to fourteen days. The filariæ which were present in seventeen, and eighteen-day mosquitos were not different in size or any other respect to those in thirteen-day mosquitos.

The young filariæ are found in the tissues of the thorax, in those of the head and neck, and in fewer numbers in those of the abdomen. The tissues of the head are examined by cutting through the neck with a sharp knife and placing the head, with the proboscis attached, on a separate slide. By carefully dissecting with needles the tissues of the head, and separating the parts of the proboscis, two or three filariæ will almost invariably be found in this situation, and I have lately on two occasions found a filaria lying stretched out lengthwise partly within the tissues of the labrum of the proboscis, the remainder of its body being curled up in the tissues of the head. Without dissecting up the tissues of the labrum these filariæ could be plainly seen with a $\frac{1}{6}$ inch objective through its fairly transparent tissues, indulging in sinuous undulatory movements, and a very little manipulation with the needles sufficed to free the filariæ, when their movements changed from the snake-like undulatory character to the vigorous, purposeless lashing and twisting which are characteristic of the final stage of the metamorphosis of the parasite in the mosquito.

Once having seen these large and vigorous filariæ actively moving in this situation, one can scarcely fail to agree with Dr. Bancroft that "young filariæ may gain entrance to the human host whilst mosquitos bearing them are in the act of biting," and I take this opportunity of correcting the contrary view which I expressed in the *Indian Medical Gazette* for May, 1900, which was written before I had begun to examine the heads and the proboscis of my filaria-bearing mosquitos.

The young filariæ in their final stage are from $\frac{1}{74}$ to $\frac{1}{78}$ of an inch in length and $\frac{1}{800}$ of an inch in the greatest breadth, and they can be fairly easily seen with the naked eye) (or better with a small magnifying glass), as they twist and wriggle about on the slide. They taper towards the head and towards the tail, and the latter is provided with three projections which can be spread out or drawn closely together in the animal's movements. The head end is rounded, and the mouth, which is very extensile, can be pushed out to form a little cone-like projection which sways from side to side and is drawn in and pushed out as if searching for food.

The filariæ are provided with an alimentary canal, which, at a somewhat earlier stage, can be seen to be very freely movable within the animal's body, and to be of varying shape in the different parts of its course. Near the anus it is wide and then narrows quickly to open at a short distance from the tail. On each side of the alimentary canal near the head, and again at a point about the middle of the body, the protoplasm is differentiated into other organs —probably reproductive organs.

In the tissues of the mosquito the filariæ can, I believe, move about by boring their way from one place to another; for on a slide filariæ, which are almost completely imbedded in a piece of tissue, can be seen to gradually protrude themselves from the tissue until they are quite free. In water, however, their powers of locomotion are very limited, but though they do not change their position on the slide, they indulge in the most vigorous lashing and twisting movements incessantly. If left in water for some time these movements gradually slow down, and in from two to three hours the filariæ die. The following experiment, however, would appear to show that in serum or blood they find a more congenial medium :—

A mosquito (Anopheles Rossii) which had fed on filariated blood on May 11, contained the final stage of the filariæ on May 24. These filariæ were first examined in water, and after they had been on the slide for over an hour their movements began to slow down. I succeeded in moving one to the other end of the slide without injuring it, and having pricked my finger, introduced the blood beneath the cover glass, and surrounded it with vaseline. The movements of the filaria at once became more active, and it tossed the corpuscles about in every direction. The filariæ that were in water alone died in two hours and a half, but this one continued vigorously active for over six hours. Its movements then became slower, and it died in seven hours.

If this experiment can be confirmed, it will, I think, afford a strong proof in favour of the suggestion that the filariæ pass directly from the mosquito to man without first having to exist for some time in water.

The object of the experiments described above was not so much to study the minute changes which occur during the metamorphosis of the filaria, as to prove, by obtaining on repeated occasions the final stage of the parasite in Anopheles mosquitos, that members of this genus of mosquito are capable of acting as efficient intermediary hosts, and I believe that, in Travancore at least, the Anopheles Rossii, and at least one other species of this genus, are the usual intermediary hosts of the filaria nocturna. I have, however, copied from my notes a few diagrams which differ from, or are not figured in, Manson's original drawings. The notes attached to them are sufficiently explanatory.

I have not, as a rule, examined my mosquitos at a sufficiently early stage to form an opinion as to whether a "casting of the sheath" occurs prior to the migration of the parasite to the tissues of the body. A fine "integument," continuous with the characteristic tail, and completely encircling the body, is, however, visible from an early stage of the development of the parasite; and in the later stages, after the filaria has been immersed in water for a time, this integument forms a perfect "sheath" surrounding the animal. Had Dr. Manson not definitely stated that a "casting of the sheath" occurred, one would have probably considered this integument to be the original sheath. The escape of granular matter from the anus is not due to pressure of the cover glass, as suggested by Dr. Bancroft. It occurred almost invariably in the "sausage-shaped" stages of my filariæ, which were examined without applying a cover-glass.

I do not consider that the filariæ specially select the muscles of the thorax to migrate to. The fact that so many more are found developing in the thoracic tissues may be due to the ease with which they can pass from the crop—which is a pouch of the œsophagus lying in the thorax and serving as a reservoir for food,[1] and from the œsophagus into the thoracic muscles, before they are acted upon by the digestive juices. The majority of the filariæ which reach the stomach are probably digested; a few, however, succeed in migrating into the abdominal tissues and undergo the metamorphosis there just as those in the thorax do.

Discussion.

I. Dr. Patrick Manson said that this important paper by Major James, coming on the top of a recent paper in the Journal by Dr. George Low, of the London School of Tropical Medicine, may give rise to questions as regards priority of discovery. I am in a position to state that both observers were working independently, that their observations were made practically simultaneously, that one confirms the other, and that therefore both share the merit of making an important contribution to tropical medicine. The observations I made on the life history of the filaria were made twenty years ago, and before the duration of the

[1] Woldert, *Journal of the American Medical Association*, February 10, 1900.

life of the mosquito had been ascertained. I took the description current in books on natural history at that time, assigning a limit of about one week to the life of the mosquito as correct. I now know this to be wrong. My feeling is that Bancroft is approximately correct when he states that a period of about three weeks is necessary to the completion of the metamorphosis of a filaria in the mosquito. Still it is quite possible that in particular species of mosquitos and in the presence of high atmospheric temperature the process, as Major James suggests, may be completed in the time I originally assigned to it. I do not think so, but it is possible. We know that temperature has an important influence on the development of the malaria parasite in the same class of insects. The fact of the presence of the filaria in the proboscis of the mosquito suggests, without actually proving, that the parasite is directly inoculated into man by mosquito bite. At the same time we must remember that the fully metamorphosed filaria is sometimes found around the stomach, about the viscera, and elsewhere than in the head of the mosquito. It is quite possible, therefore, that it may leave the insect by some other channel than the proboscis, and that it may be passed into water with the fæces, the eggs, or even by the proboscis. We know the young filaria can live for several hours in water. It dies therein after a time, possibly from want of food. It may be that, as the embryo of dracunculus, it would live longer in dirty water—that is, water containing food. Experiments to test this should be made. If anyone is foolhardy enough to submit to be bitten by filariated mosquitos, and if subsequently no young filariæ be found in the blood, it must not be concluded from this that mosquito bite is not the medium of infection. My belief is that before embryos can be found in the blood by ordinary microscopic observation, large numbers of parent filariæ must be present in the lymphatics. In many cases we know that hundreds of parent filariæ are present. Thus in one case only two or three embryo filariæ are found in each drop of blood; in other instances as many as 600 or more are found in a drop, that is, 300 times as many, implying the presence of 300 times as many parental worms. Assuming that one male and one female produce an infection of two embryos to the drop, there must be 600 parents to produce 600 embryos to the drop. It is by no means certain that two embryos per drop is the minimum of infection; if the minimum infection is less than this, we may infer that to produce a recognisable infection by mosquito bite it may require dozens of inoculations, on the assumption that the filaria is conveyed by mosquito bite. One can easily understand how it is that high degrees of filarial infection occur in natives, and how rarely filarial infection is detectable in Europeans. The native sleeping constantly in the same house without a mosquito net is month after month, and year after year, infected by his own filaria-charged mosquitos, whereas the European, though he may be bitten by filariated mosquitos once or twice, is less likely, in consequence of his superior hygienic surroundings, and his use of the mosquito net, to be repeatedly and richly infected. It is much the same in this respect with the filaria as with the malaria parasite. The subject of filariasis is by no means worked out. It is of great importance on its own account, but even more on account of the remarkable analogy that obtains between it and malaria. The analogy runs through the entire histories of both parasites, extending even to the pathological effects. Both are removed from the human blood by the mosquito, both develop in the mosquito's tissues, both probably quit the mosquito *viâ* the proboscis, both are inoculated by mosquito bite, both exhibit a remarkable periodicity in the human blood, and both give rise to recurring fevers. The further study of the filaria is therefore highly desirable as being calculated to throw yet additional light on the more important malaria parasite.

II. Major Ronald Ross, I.M.S., referred to the case of an Englishman living in the West Indies, a patient of his, who suffered from elephantiasis of the right leg, and who had long ascribed the disease to the bite of a mosquito on the right ankle.

III. Mr. D. C. Rees brought forward a fact tending to prove that some species of mosquito serve better as hosts for the filaria than others. He said: "I have recently infected a number of Culex tæniatus with filaria nocturna. Unfortunately they all died by the twelfth day. On examining, the filaria were all observed in the thoracic muscle of the mosquitos, but in a very immature condition, and at about the stage found in Bancroft's mosquitos at the sixth day. The temperature at which I kept these mosquitos was 80° F., which seemed favourable to them, for they bred freely. Both Bancroft and James seem disinclined to believe that filaria nocturna casts its sheaths in the mosquito's stomach. I think there can be little doubt it does do so. We have specimens in the London School of Tropical Medicine of the blood exuded from the stomach of the mosquito, which show that ecdysis has taken place, for the filaria and its sheath can be seen side by side.

NOTE ON THE ETIOLOGY OF FILARIASIS.

By Lieut.-Col. J. Maitland, M.D.,
Professor of Surgery, Madras Medical College.

Without desiring in any way to attempt to minimise the importance of the remarkable observations made by Mr. G. C. Low, and published in the *British Medical Journal* of June 16 last, it is necessary to point out that all the information that has hitherto been gathered from clinical research appears to be opposed to the correctness of the theory propounded by him. There can be no doubt that the phenomena observed by Mr. Low afford strong presumptive evidence in favour of a theory of direct inoculation, but on the other hand, many well-attested facts afford evidence of an opposite character.

One of the most important items of evidence opposed to the inoculation theory lies in the well-known fact of the comparative immunity to the disease enjoyed by the white races. It is true that in certain isolated localities (the Friendly Islands is one, I believe) a certain proportion of the white population have suffered, but such instances are very rare. In the littoral districts of India, where filariasis is extremely common, and where a very large proportion of the natives and a considerable number of half castes become affected, it is extremely rare for Europeans to acquire the disease. The very few cases that are met with amongst the latter race are generally found amongst the poor classes, and the victims are usually so-called "country-born" Europeans, that is, Europeans born and bred in India. After an extensive experience of this disease, lasting about twenty-four years, I have never met with or heard of a case in a well-to-do European in India. If the inoculation theory be correct, what is the explanation of the extraordinary immunity enjoyed by Europeans, especially those who belong to the well-to-do classes? It is true that the latter usually, but by no means universally, sleep under the protection of a mosquito net or a punkah, yet between the hour of sunset and the time of retiring to rest the European is freely exposed to the bites of mosquitos. During the times that mosquitos are plentiful probably few, if any, Europeans escape being vigorously attacked by these insects; indeed, they appear to attack the white man with greater avidity than they do those with dark skins. Then again the punkah, under which many Europeans sleep, being, as it generally is, propelled by human agency, is but a poor protection against mosquito bites. Yet in places like Madras we find white people, unaffected by filariasis, living in close proximity to native houses where infected persons reside; in fact, it is not uncommon to find native servants suffering from the disease residing in the same houses as their masters and mistresses.

The fact that the embryo filariæ do not enter the general circulation in any number until past midnight does not

affect the theory of inoculation one way or the other. If it be true that the worm enters the human body directly from the mosquito's proboscis, the presumption is that the parasite is always prepared for immediate action the moment that the mosquito commences its suctorial operations. If the worm were not thus prepared, its opportunity of escape would in all probability be lost, as the time occupied by the mosquito in completing its meal is a comparatively short one.

In connection with this question of the immunity of white people to filariasis it is a noteworthy fact, and one which deserves the attention of those engaged in the study of malaria, that well-to-do Europeans in Madras also enjoy an extraordinary immunity against malarial fevers as compared with natives. A very large proportion of the European residents of Madras never suffer from malaria, yet the disease is very prevalent amongst the native population.

In connection with this subject certain observations made by Captain S. P. James, of the Madras Medical Service, and published in the *Indian Medical Gazette* of May last, are of interest. Captain James, for reasons detailed in his paper, states his belief that "the Anopheles mosquitos, rather than the Culex mosquitos, are the usual efficient hosts of the parasites." He admits, however (and it is a matter concerning which there is no doubt), that the Culex does frequently act as the intermediary host. I have myself followed the process of metamorphosis of the filaria in great numbers of mosquitos, and have always used the Culex as the host in these experiments.

Water has hitherto been considered to be the probable medium by means of which the parasite reaches the interior of the human host, and there are several arguments in favour of this assumption. It has frequently been noticed that certain communities, having a common supply of drinking water, have suffered severely from the disease, whereas neighbouring communities, obtaining their drinking water from a different source, have suffered to a much less severe extent.

A notable example of this occurs in the neighbourhood of Madras, where a very large proportion of the inhabitants of a certain village are affected with filariasis, whilst those who reside in the adjacent part of the city itself suffer to a very much smaller degree. The inhabitants of the latter district obtain their drinking water from the supply common to the whole city of Madras, whereas the inhabitants of the village depend upon water obtained from shallow wells. One of these wells is found at the back of nearly every house in the village, and in the evenings mosquitos may be seen in great numbers flying to and fro between the interior of the houses and the adjacent wells.

Water derived from shallow wells and pools is believed to be a much more fertile source of the disease than that which is derived from rivers or running streams, or conveyed by means of pipes from large reservoirs. The immunity enjoyed by the European in India is believed to be due to his drinking only boiled or filtered water. A very interesting circumstance in connection with this question came under my notice a short time ago. Four Eurasian lads who took to bathing in and drinking the water from a certain pond, all became simultaneously affected with filariasis, and from one of them, who came into hospital, I succeeded in removing several mature worms.

It must be remembered that, so far as Mr. Low's observations have gone, there is nothing in them to negative the theory that filariasis is a waterborne disease. It is quite possible that when the young filaria reaches the mosquito's proboscis it may pass from thence into the water, and not, as Mr. Low supposes, directly into the blood of human beings. Manson's belief in an intermediate aquatic existence may after all be correct.

Captain James, in the paper already alluded to, points to another fact which is opposed to the inoculation theory, namely, that persons affected with filariasis may live in intimate association with others without the latter becoming affected. If Mr. Low's theory be correct, how does it come about that any of the other persons living in the same house with a case of filarial disease escape infection? The presumption is that the whole community would become affected sooner or later. Yet many escape.

(*To be continued.*)

Reviews.

TROPICAL DISEASES: a Manual of the Diseases of Warm Climates. By Patrick Manson, C.M.G., F.R.S., LL.D.Aberd., with 114 illustrations and 2 coloured plates. Revised and enlarged edition. Cassell and Co., Ltd., London, Paris, New York and Melbourne. 1900. Pp. 684.

So rapidly has tropical pathology advanced, even since the first edition was published in 1898, that a second issue of this well-known work had become a necessity. The plan of the present edition is modelled upon the previous, but some eighty more pages have been added to the text, and a portion has been almost re-written. A small space has been devoted to the blood parasites of lower animals, and diagrams of the Drepanidium met with in frogs, the Pirosoma bigeminum of Texas fever in cattle, and the Halteridium of birds are given. The stains employed for malarial blood are more exactly enunciated than in the previous edition, and the whole *technique* connected with the microscopic diagnosis of the malarial parasite are so clearly set forth that any earnest man, in however isolated a position of the globe he may reside, can educate himself in all the details of malarial enquiry. The chapter on Yellow Fever, in a work in which all is excellent, is especially well described. It is clearly demonstrated that we have not yet, unfortunately, arrived at a decisive conclusion as to the cause of this disease, the Bacillus icteroides not being universally accepted as the toxic agent, although many believe it to be so.

Although the nature of the virus of ulcerating granuloma is not known, we have a considerable amount of fresh information upon this subject. It would seem, however, that we are dealing with a specific ailment, and although, even now, but limited information as to the prevalence of this disease is to hand, we agree with Dr. Manson that "this disease is widely distributed in the tropics."

The subject of Filaria occupies some forty pages, none too large a space to be allotted to so important a subject. The author has been able to incorporate the recent findings of Low, James, and others as to the communicability of the filarial parasite by mosquito bites, whereby a new and important advance was made in our knowledge of filarial disease.

The subject of Pinta has been further elucidated, and a diagram of the fructification of cryptogamic epiphytes in pinta is a welcome addition.

Ixodiasis, a new name in tropical literature, is a disease due to two species of acarus, the Argas persicus and Argas moubata. The former receives its name from the fact that it is met with in Persia, and the

latter, called "garrapato" by the Portuguese, is a tick frequently met with in South Africa. Although the bite of these insects is not invariably followed by serious consequences, there seems little cause to doubt their frequent power of producing a toxic influence, causing "severe pain, delirium, convulsions, and sometimes even death."

It need scarcely be said that Dr. Manson's work will be welcomed by all tropical practitioners. It is written in a clear and concise style, and it is well illustrated. No disease is dealt with in a fashion so elaborate as to hamper the space devoted to others, and we have to thank the author for placing before us a book which is thoroughly up to date. The care and trouble necessary to produce a work of this kind is immense, and the medical profession and all tropical residents are under a deep debt of gratitude to Dr. Manson for so excellent a guide to tropical diseases.

New Remedies, &c.

SPHAGNOL SOAP. — This soap is a production of British peat, and if the genuineness of the soap is to be judged from the odour of the tablets, one has no hesitation in declaring it genuine. The peat smell is pronounced, and as it is impossible to exactly imitate it by chemical products, the fact that the salts and extracts of peat are used in the manufacture seems conclusive. The odour will remind the Irishman of his potheen, the Scotchman of "peat reek," and the Englishman of pleasant days spent in the neighbourhood of both. The soap in use is pleasant, soft, soothing, and cleanly. The active principles are said to be creasote and sulphur; but in a substance such as peat, rich in a plethora of earthy salts, the ingredients are legion. Peat softens water. A Scottish mountain stream in flood, brown from passing through peat, is always welcome to the housewife as a ready means of washing and saving soap. From experience of the soap we have formed a favourable opinion of it.

THE "DYLISSIA" MEDICAL CREAM, manufactured and issued by Durant & Co., Rosebery Avenue, London, is an effective application in allaying the irritation produced by "prickly heat," the "dry skin" of cold weather in the tropics, and the annoyance caused by Dhobbie itch. It will be a welcome addition to the means of coping with numerous cutaneous ailments peculiar to the Tropics.

Correspondence.

To the Editors of the "Journal of Tropical Medicine."

DEAR SIRS,—In your leading article of April, 1900, on "Small-pox in the Philippines," while speaking of the supply of vaccine to the Far East, you omit to mention what I believe to be the chief source of supply of vaccine, namely, the Shanghai Vaccine Station, which is a branch of the Shanghai Municipal Health Department. This institution is now in the third year of its establishment, sends vaccine throughout China and a constant supply to the Philippines, Malay Peninsula, Japan, and Russian Asia.

I am, yours faithfully,
ARTHUR STANLEY, M.D., B.S.Lond., D.P.H.,
Health Officer of Shanghai and Director of the Shanghai Vaccine Station and Bacteriological Institute.

May 20, 1900.

To the Editors of the "Journal of Tropical Medicine."

DEAR SIRS,—I imagine the "pipsas" of Sir Joseph Fayrer, referred to in Journal of June, are what are known in this district as "Dam Dooms." The origin of the word I am ignorant of. The "Dam Doom," in appearance, is very like the ordinary house-fly on a slightly smaller scale; its head bears a curious resemblance to that of an elephant. They live amongst the jungles, along the lower ranges of the Northern Himalayas, and are found in greatest numbers in the vicinity of running water. The cold weather is the only time they are seen, and are most active in the evening; as cultivation extends they disappear. The face is seldom bitten; the hands and feet, if exposed, are the situations of attack. The bite is not felt, but a few hours afterwards the itching is intense — in some people each puncture becomes an ulcer. There is no mistaking the bite—a subcutaneous hæmorrhage about the size of the head of an ordinary pin. If this is promptly opened and sucked, no ill effects are produced. To avoid the attacks of these pests coolies wear trousers, the hill tribes finding safety in their habitual state of filthiness.

Joyhing, North Lakhimpur, Upper Assam,
July 20, 1900.

Yours faithfully,
W. RUSSELL.

News and Notes.

WE learn that at the Paris Exhibition two "Grands Prix" were awarded to the Pasteur filter, one to Monsieur Chamberland in the class of General Hygiene, and one to the manufacturers in the class of Military Hygiene, and that no "Grand Prix" was awarded to any other filter whatever.

In view of the large number of filters exhibited, and of the interest and importance which attaches to the question of filtration at the present time for military as well as for general purposes, we are pleased that an English firm of the well-deserved repute of Messrs. J. Defries & Sons, Ltd., has carried off the honours.

THE ANGLO-AMERICAN HOSPITAL SHIP "MAINE" has proceeded to China, and will serve as a Station Hospital Ship or a transport for sick and wounded as occasion may serve.

A MOSQUITO LAMP.—Messrs. Montague, of 101, New Bond Street, London, have brought out a mosquito destroyer. It is a reproduction of the "Swatow" lamp used in China, but curiously enough it is unknown elsewhere. The lamp is a most effective means of killing mosquitos, more especially when they gain entrance beneath the mosquito nets. The lamp is well known in Southern China and extensively used. Mr. Cantlie exhibited the lamp and explained its action at the Tropical Section of the British Medical Association at Ipswich.

LEPROSY IN THE PHILIPPINES.—The U.S. Government have decided to segregate the lepers in the Philippines by sending them to a suitable island. Leprosy is reported to have been introduced into the Philippines in consequence of some 150 lepers being sent thither in the year 1633 from Japan. Since then leprosy has spread, until at the present moment it is estimated that there are 30,000 lepers in the Archipelago.

CASES OF BERI-BERI IN THE EDINBURGH ROYAL INFIRMARY.—Dr. T. O. Affleck has had six cases of beri-beri under his care in Edinburgh. The patients had all travelled and resided in warm climates. Sodium bromide and nux vomica were the drugs used in treatment, and under a generous diet the patients all did well. Bacteriological examination of the blood gave negative results.

THE SIGNIFICANCE OF THE BACILLUS COLI COMMUNIS IN DRINKING-WATER.—In an article contributed by Drs. Linsley and Stone to the *Medical Record*, September 1, 1900, upon this subject, the conclusions arrived at are as follows:—"From the consideration of all these facts we are driven to conclude that waters in which the bacilli coli communis are found to exist for any length of time are dangerous to the public health, and that the only safe course is to condemn all such. An example proving this is the recent outbreak of typhoid fever in West Burke. From the first there seemed good reason for suspecting the water as the source of the trouble, and samples were sent to the laboratory and examined. The cases in this town were undoubtedly typhoid, but no typhoid bacilli were found in the water. It was, however, found infected with the bacillus coli communis. If the water had been examined before the outbreak and found contaminated, as it undoubtedly was, the trouble might have been averted."

Letters, Communications, &c., have been received from:—

B.—Dr. R. C. Bennett (Trinidad).
C.—Mr. Chapman (Birkenhead).
D.—Dr. A. B. Dalgetty (Madabpore).
E.—Dr. Wm. Elliott (Crowborough).
F.—Dr. R. Felkin (London).
H.—Staff-Surg. D. Hoskyn (Channel Squadron).
L.—Dr. W. P. F. Law (British Guiana).
M.—Dr. M. Macnicol (Bengal).
O.—Dr. W. Osler (New York).
R.—Dr. W. Russell (Upper Assam).
T.—Dr. Chas. Todd (Islington).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

Notices to Correspondents.

1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

CONTENTS.—OCTOBER, 1900.

Original Communications.

OBSERVATIONS OF MOSQUITOES.

By P. W. Bassett-Smith, Staff Surgeon, R.N., F.Z.S.

I.

THE DIAGNOSIS OF MOSQUITOES.

Published by Permission of the Admiralty.

During the past year there has been a remarkable influx of literature on the life history, habits, and structure of mosquitoes and their near allies, the midges, particularly with regard to their relation in the dissemination of the malarial parasite.

The very full and interesting compilation of Major Giles, I.M.S., "Gnats and Mosquitoes," leaving but little more to be done; also the smaller but extremely useful publication, "Mosquitoes and Malaria," by Christy, which goes deeply into the subject as far as is possible with our present knowledge. Both these authors naturally point out the great differences in the structure of the head and its appendages as means of differentiating the sexes and genera, the parts which are so intimately connected with the ingestion and ejection of the malarial and filarial parasites.

I would here briefly draw attention to the great distinguishing characters of the generative and caudal appendages of these insects, showing how easy it is to readily recognise the sex, in cases of doubt or mutilation, by an examination of these parts, which the annexed photographs show clearly. (*See plate.*)

Giles, on page 27 of his work, states that "the ninth abdominal segment is quite invisible in the usual position of the insect, showing only two lobed appendages thickly coated with hairs and scales, and not showing any very prominent difference in the two sexes." It is this last statement that I would take exception to, and consider to be misleading, as the differences are very marked.

In the female *Culex* and *Anopheles* there are several small hairy plates and two large foliaceous terminal ones, the latter being covered with rather long hairs or setæ. These plates are placed dorsally, having their upper borders close together, spreading below like the gables of a roof, under which the eggs pass to be further guided by the hind legs on to the surface of the water.

In the male *Culex* these parts are much more differentiated, forming sexual organs which act as claspers (*hypopygium*), but varying very much in appearance in the different species; between these is placed the penis as described by Giles.

The proximal joint of the clasper is always, so far as I have seen, provided with very long and numerous hairs on the outer border, but on the inner surface is found a specialised sex organ in the form of collections of very delicate hairs, distinctly grouped, which are probably connected intimately with nerve filaments and have true sexual functions. The distal joints vary considerably; for instance, in a light brown *Culex* from China, it is typically claw-like, being narrow and sickle-shaped, sharply bent on the proximal joint; on the concave surface a series of pectinate or comb-like processes are seen, which are very peculiar and quite distinctive. In another species, also from China, commonly known as the "small tiger mosquito," the terminal joint of the hypopygium is very slender or stilt like, slightly enlarged at the extremity, where a few very small delicate hairs are to be made out; but besides these there is a single style-like prolongation placed at right angles to the joint, being about three times its breadth at this part.

If the caudal extremity of a male "midge," *Chironomus plumosus*, be now compared, a striking difference is seen; the hypopygium consists of a single, somewhat concave, elongated joint with very long hairs on the outer border, also a short parallel row of minute ones placed near the extremity on the inner side; on either side of the penis, two short hairy lamellar plates are also present, as well as accessory spines or claws at the base of the clasper.

In some *Culex* larvæ sent to me from Bombay, probably after the first month, the extraordinary length of the respiratory tube is very remarkable, being two-fifths as long as that of the whole animal; this progressively shortens at each moult, but in the final stage, as is well known, it is still very conspicuous, both it and the character of the "tail plates" being very different from those of the *Anopheles* larva.

The head portion of the young larva is proportionately very large as compared with the thoracic.

II.

The Habits of Mosquitoes—Observations at Portsmouth.

When the expedition for the study of malaria, under the direction of Major Donald Ross, I.M.S., visited Sierra Leone and the West Coast of Africa in 1899, several important distinguishing points were brought to light in the diagnosis of the harmful *Anopheles* from the less dangerous *Culex*—among them the characteristic habits of the living larvæ and imago in the positions they assume when at rest; these being easily detected by others than expert entomologists, and therefore very useful to recognise in the extermination of the mosquitoes themselves.

The characteristic attitudes are now well known, being quoted in every book on the subject. It is, however, of great importance that their observations should be corroborated in various different parts of the world. Are these characters general, or are they limited to special species only found in certain localities?

Lately Christy, in his book "Mosquitoes and Malaria" (1900) has shown that these characters are also found to hold good for species of the two genera in India.

Recently I have been able to breed and study many species of *Culex* and also *Anopheles Claviger* found at Portsmouth. The first *Anopheles* I found at the end of August, a single female among a great number of *Culex* of both sexes, obtained in the garden among some thick ivy bushes. Since then I have taken many more of the former genus of both sexes; but it was not for some time that the breeding place could be discovered. This was a small shallow stone tank in the kitchen garden supplied by a tap, the water being turned on occasionally: in the tank was a good deal of weed, the water looking greenish and turbid; it contained great numbers of *Daphnia*, *Cyclops*, &c., with abundant eggs, larvæ, and pupæ forms of both *Anopheles* and *Culex*.

The larvæ of the *Anopheles* were by their horizontal position and the anatomy of the tail unmistakable, conforming in habits with those found abroad; the resultant imago also showed the typical position when at rest, more marked, however, in the female than in the male.

I have not so far seen published the fact that outside the tropics these readily-recognised diagnostic points have been observed, though Giles, in his book, "Gnats and Mosquitoes," describes them as characteristic of the genera; if, however, we can get many observers to record the same, then they will become real generic distinctions. Peculiarities of habits in the *living* organisms will take, as they ought to in all branches of natural history thus studied, that position of importance which will render such investigations of double the interest to the general public that the mere laboratory anatomical detail furnishes.

A further point of great interest is the fact that here both genera, *Anopheles* and *Culex*, were breeding in the same tank near to a house; they therefore would come under the heading of "urban and domestic" rather than "rural and wild."

Both *Anopheles* and *Culex* when on the wing hum.

ANCHYLOSTOMIASIS IN THE LEEWARD ISLANDS.

By H. A. Alford Nicholls, C.M.G., M.D., F.L.S.

My paper under the above heading in the May number of The Journal of Tropical Medicine has drawn forth some remarks from Mr. William M. McDonald, the late acting Medical Superintendent the Holberton Institution in Antigua. Mr. McDonald's remarks and assertions do not necessitate any serious attention from me, for they leave the facts I brought to the attention of the Government of the Leeward Islands uncontroverted. But as silence on my part may be misconstrued, I forward this second paper for publication in the Journal.

Mr. McDonald's official letter or report, which he erroneously describes as an official despatch, was written six weeks after he had diagnosed anchylostomiasis, and after he had had experience of some twenty cases of the disease in a large hospital, to which the worst cases of disease amongst the labouring indigenous population are naturally sent. His letter was not written in the calm and cautious way usually adopted by scientific men, but was couched in language that had a tendency to alarm unprofessional authorities, and that would, doubtless, have created a panic amongst the people had it been published. As one of the senior members of the profession in the Colony, it became my duty, therefore, in the report I was called on to make, to allay any undue alarm that had arisen; and, in doing so, I alluded to my young *confrère* with courtesy, and gave him all the credit that was his due. And Mr. McDonald's paper in the Journal, if anything, emphasises the fact that my Report to the Government was advisable and timely.

In my report on the whole question, which is the paper you published, I was careful to give the hitherto undisputed authority of such men as Leonard Rogers, Dobson, Bilharz, and McConnell, for some of the

statements I made, but Mr. McDonald now, in effect, asks the members of the profession practising in the tropics to set aside the rich experiences and careful teaching of these men, and to accept his limited experience and alarmist theories in lieu thereof. These authorities teach that a moderate infection with anchylostomata may do no harm—one of them, Dr. Dobson, shows that over 80 per cent. of the healthy inhabitants of most parts of India, who are certainly not physically inferior to the Antiguans, harbour the parasite in numbers varying from units to hundreds. And, another of them, Dr. Leonard Rogers, found a moderate infection in 66 per cent. of fifty healthy men in Assam. Whereas, Mr. McDonald gravely asserts, "I say most definitely that the harmfulness of a moderate infection cannot be overestimated." Further comment on this point is needless.

There is nothing in my Report to warrant the statement contained in the second paragraph of Mr. McDonald's paper. I said, "Facts that have come within my knowledge have satisfied me that the presence of a comparatively small number of anchylostomata has been set down as the cause of an anæmia due to the ravages of the malarial parasite in the blood." In writing this paragraph I had no thought of Mr. McDonald or his practice in my mind; but, on referring to his report, I find that he has confused an infection with the parasite and the result of repeated infections, *i.e.*, anchylostomiasis. For, in his Report, he says of anchylostomiasis, "In some countries it is a positive curse; in Egypt 75 per cent. of the people have it." It is asserted that in Egypt 75 per cent. of the population harbour the worm, often harmlessly, but it will be news to medical men in that interesting country to learn that three out of every four people there are suffering from anchylostomiasis.

Mr. McDonald is good enough to say that I am "ignorant of the condition of affairs in Antigua." This statement simply shows that he has not carefully studied the history of sanitary affairs of what is, I believe, his native country as well as the place of his professional work. Nine years ago, before Mr. McDonald's student days, I was sent on a mission by the Home Government to Antigua, as well as many other islands in the West Indies, to inquire into certain matters that necessitated me giving much attention to the whole medical history of the island, and that required me to investigate the condition of the town and country districts. And, moreover, for a considerable time I was engaged in a pathological investigation in the Government laboratory, then placed at my disposal by the local Government for the purpose. In my Report to the Secretary of State, which was published as a Blue Book, I had much to say concerning Antigua and the sanitary condition of the island. That condition is worse now than it was then for the simple reason that the decadence of the sugar industry, which is practically the only one in the island, has thrown the people into abject poverty, and rendered the Government dependent on imperial assistance "to pay its way," in spite of rigid economy in all departments, including the medical service. In the circumstances, therefore, Mr. McDonald's excellent suggestion to supply water by means of pipes, &c., to every range of labourers' houses on each estate and in every village, is an utterly impossible one on account of the enormous expenditure it would entail. Already within the last ten years or more the Government have spent large sums raised by loan in attempts to improve the water supply, and in other ways the Government has done everything possible with the means at their disposal to mitigate suffering and to lessen the high mortality. Years ago, when the island was rich and the population small, sanitary questions were disregarded, as they were in those days in other places at a far less distance from the heart of the empire. Nowadays the desire for sanitary reform exists but the means are absent. Yet, even now, the sanitary condition of Antigua, bad as it is, is not so bad as other places I have had to visit officially in the West Indies; and, according to published accounts, it is infinitely better than many other places in the tropics.

As regards anchylostomiasis in these West Indian islands much can be done by the medical men themselves to arrest its prevalence. The West Indian people are not so ignorant as they are often made out to be, and they have a keen appreciation of anything that is for their benefit—when they are quite satisfied that it is really for their benefit. A case in point occurred in my practice here a few months ago. A young man was sent to me from a distant country district as he was very ill. I found him to be suffering from severe anchylostomiasis, and his stools were loaded with ova of the parasite. He was cured and sent home, when his younger brother and their father came in suffering also severely from anchylostomiasis. They were cured in their turn, and I found out that their house had no privy, and that the night soil was thrown away on the land in the neighbourhood. I took particular care to explain to these people the nature of the disease for which they suffered and what arrangements were necessary to be made to prevent re-infections. They listened carefully to me, and thanked me profusely for my advice, which they said they would not only follow themselves, but would try to get followed by all their neighbours. In the smaller West Indian islands, owing to causes that have their roots in slavery times, it is too often the practice to throw the responsibility of anything and everything on the Government, and to look to the "powers that be" to arrange matters that the people should do for themselves. As I said at the conclusion of my first paper, "obvious and simple sanitary measures are all that are necessary to arrest the propagation of the parasite; and, therefore, to rid any district of anchylostomiasis." In a country, such as Antigua, where, owing to economic causes an efficient and therefore very costly water supply is at present impossible, and where the people are Christianised and fairly intelligent, such obvious and simple measures should be easily carried out within a reasonable time, and meanwhile the cases of anchylostomiasis when they crop up can be treated at the hospital, or by the resident medical officers of the districts into which the island is subdivided.

THE ÆTIOLOGY, SYMPTOMS, DIAGNOSIS AND TREATMENT OF ROUND-WORM INFECTION.

By J. Preston Maxwell, M.B., B.S., F.R.C.S.
Changpoo, South China.

I. Prevalence and Ætiology.

In England the importance of infection of the intestinal tract with the round worm is hardly recognised, owing to the comparative rarity of the disease, but in the Changpoo valley, South China, where I am working, the case is very different. I believe that without any exaggeration 99 per cent. of the inhabitants are infected with this parasite at some time or other during their life. And it is the exception to find a child over the age of two who does not respond to a dose of santonin by the passage of one or more worms. On the other hand, tape worms and thread worms are very uncommon. To prove my assertion, let me cite the case of the girls' and boys' boarding-schools in Changpoo. In the former, at the time of my experiment, there were fifty-five persons, viz., the matron, two other women and fifty girls, the ages of the latter ranging from 19 to about 6. Each of these had two doses of a grain of santonin mixed with a grain of hydrarg. and cret., the first dose being given at night and the next on the following morning. With the exception of one girl who was constipated at the time, and had previously passed worms, every one of these individuals passed from one to six worms, and one girl passed fourteen. In the latter case the forty-five boys then in residence were all dosed with a similar powder, with the result that every one of them passed one or more worms. Having the subject thus thrust upon my notice, I proceeded to try and discover the mode of infection with the parasite, and a well-known fact at once obtruded itself on my notice. The Chinese here are in the habit of manuring some of their vegetables, not only with human urine, but with the contents of the fæcal pits which form the latrine system of China. Pursuing this question, I found that it was only certain vegetables that were manured with fæces, and many of these were only manured thus in their earlier stages. And I also found that only a few of these vegetables were eaten raw, most of them being well cooked. The latter could be excluded from my enquiry, as it was certain that the cooking would destroy the parasite if present. So attention was directed to three or four vegetables, viz., garlic, two forms of leek and onion. The last named is very rarely eaten raw, and may on that ground be excluded. Obtaining some garlic from the fields, I was fortunate enough to find in a washing from the portion between the ground and the leaf—it is this portion which is eaten—not only the empty round-worm ova, but the actual minute embryos moving about under the field of the microscope.

The researches of many observers have proved the exceeding power of resistance of the round-worm ovum, and it has been artificially cultivated up to the stage of the embryo in the ovum. It has also been proved that if ova in this condition are swallowed, the embryos are set free in the stomach or upper portion of the intestinal canal, and infect the individual. I was able to confirm the researches of Leukart as to the change in form of the round-worm ovum, which, as it develops, loses its rugged character and becomes smooth on the outside. And while not denying the fact that the ingestion of the ova in this condition is sufficient, I would suggest that in many cases at least the actual embryos are swallowed, having escaped from the ova before ingestion. On subsequent occasions I was able to confirm the truth of my first find. On some occasions I found only empty ova, and on others the free embryos, while on certain occasions I found nothing. This was what was to be expected. Before use the Chinese wash these vegetables, and if well washed and not manured for some time before gathering, it is easy to conceive that nothing may be found. After the first occasion I always bought my vegetables from the stalls in the streets, so as to examine the actual article ready for food. And the crowning touch was afforded me when I discovered the round-worm empty ova on a piece of leek taken from a small boy's mouth in my consulting room.

Having thus settled a possible mode of infection, the next question came to be, was the eating of garlic and leek (of which there are two kinds) sufficiently general to warrant my calling *them* the means of carrying the infection? For several months I investigated this question. I have never yet caused a baby who had only fed on milk (human), rice cakes and sugar cakes, to pass worms. And in only about three out of 150 cases could the eating of leek or garlic be excluded. In these three cases it is possible that sugar cane was the offender. This, in the early stages, is manured with fæces, and these three children had all eaten of it, pulled up from the ground by their own hands, and without any cleansing whatever. So that I think it may fairly be said that in the majority of cases leek or garlic is the offender. As to whether the ovum or the actual embyro is ingested, it is probably a matter of little importance.

As to the number of round worms which may be passed at a single evacuation, the largest number I have myself counted was eighty-seven, and the patient was a man aged 54. Eighty-five to ninety is by no means a rare number, and one mother told me in answer to the question "How many?" that her son, a boy of 8, had had four large evacuations composed of worms.

It is quite unnecessary for me to enter into a detailed description of the male and female worm; the text-book descriptions are accurate and ample. As to its normal habitat in the human intestine, the usual statement is that it lives in the upper part of the small intestine. Certainly it can also live in the stomach and large intestine, and vomiting of one or several worms is by no means an uncommon event. It has been withdrawn by the fingers from the pharynx, its presence there having been indicated by a tickling sensation. Sometimes the worm is ejected through the nose, and it has been observed by others that in a fatal case of malarial fever vomiting and ejection of worms from the nose is often a sign of impending death. Many cases have also been recorded of migration of the worms from the intestine to other parts of the body. It has been found free in the abdominal cavity of persons seized with acute

peritonitis, and in which it was probably a prime factor, the persons being otherwise in good health. It has been withdrawn from the cavities of abscesses in different situations.

In one case, hitherto unpublished, which has come under my notice, the dead body of a round worm was removed from an abscess pointing over the lower portion of the sternum, the patient making a good recovery, without any symptoms indicating trouble in the gastro-intestinal tract. The above case occurred in a native, and the abscess was opened by my colleague in the Changpoo Hospital.

In another case of obliteration of the vagina after child-birth, on which I operated in order to form a new vagina, it is just possible that I may have pricked the wall of the bowel high up. There was no sign of any connection, however, between the new vagina and bowel till the third day, when I found a round worm wandering about the top of the vagina above my packing, and discovered that a small recto-vaginal fistula had formed high up. The following day another worm was found in the vagina. Both worms had, undoubtedly, come through the fistula, which speedily closed.

Operative measures on a worm-infected patient appear to disturb the parasites. In a patient, aged 10, on whom I had performed a radical cure of an inguinal hernia with retained testicle, the temperature rose after operation and persistently refused to fall till I had cleared the intestinal tract by santonin. He had been purged in the ordinary way previous to operation, and the wound healed quickly by first intention without the slightest sign of inflammation. I have frequently noted the same occurrence in others of my surgical patients.

It is also a frequent event for patients recovering from chloroform to vomit one or more round worms.

Another interesting fact may be noted in this place. *Opium-smoking* in no way influences the incidence or number of the worms. I have seen a man, smoking 4 ozs. of opium extract *per diem*, pass numbers of worms, which appeared quite as lively as those from a normal patient. And opium-smokers frequently come and ask for worm medicine. *Quinine*, also, has apparently, no effect on them.

II.—Symptoms and Diagnosis.

Let us now examine the symptoms and signs of infection of the human intestine with the round worm.

I am quite aware that here I enter on debatable ground. By the cautious school I shall be considered to be romancing and credulous as regards some of my statements. Let me assure such that in my researches into the subject, I have ever kept this possibility before me, and have been careful to weigh well the possibility of symptoms and signs being referable to other causes.

Let us consider, first of all, the case of the *adult*. In this region there is a word "hùi" which means *a craving for food coming on about an hour after a good meal*. This symptom may be said to be a certain one. If a patient has got this symptom, which is a well-recognised one, the physician will find that (in this region) about 99 per cent. of such patients will pass worms under the influence of santonin. I have myself tested this in several hundred cases and found it reliable. In an adjoining region I am told that the word used is one which may be most aptly translated as "green with hunger." This is as sure a symptom as its fellow in my own region.

Another valuable sign is severe discomfort in the region of the stomach. The writer well remembers his first case of this kind. He was called one afternoon to a woman of 35, whom he found rolling about the bed and groaning, with her knees drawn up, her abdomen rigid, and an anxious look on her face. But examination of the patient found the pulse practically normal, and the rigidity only apparent and assumed. The patient complained of indescribable discomfort in the gastric region, and nausea. A dose of castor oi and santonin caused the passage of a large number of worms and the vomiting of two or three, and the discomfort disappeared as if by magic.

Since that time about twenty such cases have come under the writer's notice, and santonin has in every case worked a complete cure. In two or three cases the patients have volunteered the information that they could feel the worms moving about in their stomachs, especially after a meal. Whether this was fancy or not I am unable to say, but it is a significant coincidence that in every one of these cases santonin administered in the usual way brought on vomiting, which in its turn led to the expulsion of three to four worms by the mouth.

There is also a class of cases characterised by a marked anæmia. Its distinctive features, as distinguished from malarial and other forms of anæmia, I am unable at present to tabulate. These patients mostly complain of weakness, headache, anorexia, poor sleep, and a disinclination to do physical work. The ordinary treatment for anæmia is utterly futile in these cases, until they have been thoroughly treated with santonin and have been rid of their unwholesome guests; but as soon as this has taken place, they improve with great rapidity, their spirits rise, and life becomes once more enjoyable. And I believe this class is far larger in my region than would be deduced from the number of cases which have come under my notice.

Turning to the case of children, there are certain well marked signs which are pathognomic of the disease in question. Foremost amongst these is a large flabby protuberant abdomen, which is generally mistaken by the layman in this region for a large malarial spleen.

This sign was brought to my notice in a curious manner. Going through one of the schools of business, I was asked to see a girl who had been fairly heavily dosed with quinine for some time. I was at once struck by the fact that, although the size of the abdomen would tally with that of one containing a large malarial spleen, yet it was symmetrical, which is never found with great splenic enlargement, and on further examination I found that the spleen was not palpable, and that the abdominal enlargement was due to flatulence and a lax condition of the wall. On exhibiting santonin many round worms were passed, and the condition was much improved. On further enquiry I found that the sign was well known

to my native students, and I have verified it many times since. But only about one in ten of children infected with round worms present the sign, so that it is only of value *when present*. As to the exact pathology of the condition I am in doubt. It only slowly disappears under treatment, and I have never seen it in adults. Another symptom which is very much more common is that of gastro-intestinal disturbance. Persistent stomach ache, unconnected with the ingestion of unripe peaches and the like, ought always to suggest to the practitioner in the East the probability of round-worm infection. This stomach ache is very often combined with alternate attacks of diarrhœa and constipation, the former being perhaps the most common.

And in this case, as in the case of the anæmia of adults before noted, it is useless to prescribe drugs to cure the gastro-intestinal disturbance *before* the round worms have been expelled. In the case of children anæmia is often a marked feature, but in them it is practically always combined with signs of gastro-intestinal trouble.

Reflex symptoms are often present. Convulsions in the younger children are well known, and grinding of the teeth during sleep is also of common occurrence. Under this heading fall also three fairly common symptoms, nose picking, preputial irritation leading to manipulation of the organ and night terrors. The latter are in the majority of instances out here due to the presence of these parasites, and often cease completely on the expulsion of the worms.

Children infected with this parasite have a great tendency to sleep *on their face*, and they are also subject to perversions of appetite. Often they will gather up and eat dust off the floor or scrape plaster from the walls for the same purpose.

While speaking of appetite it must be carefully noted that infection with the round worm does not necessarily involve anorexia. In many cases it undoubtedly does, but in another class it produces the exact opposite, and I have seen a man anæmic and suffering from nothing but the effects of round worms polish off five bowls of rice and complain that he was still hungry. After evacuation of the worms his appetite markedly diminished, but his general condition improved, and his sense of emptiness disappeared.

Amongst children there is the same difference noted. Some become voracious and others have a positive disgust for food. What causes the difference I know not. Those suffering from stomach ache generally suffer from anorexia, but beyond this I cannot go.

As to the diagnosis of the disease, there is a sure and certain means in our hand. The microscope enables us to say at once, and without hesitation, whether a certain patient has or has not the parasite. A small portion of the stool is placed on a slide, covered and examined under an inch objective. If the patient is very constipated a dose of Epsom salts is an advantage. A small portion of the fluid evacuated is examined in the same way. Any ova found in the preparation are examined under a ¼-inch objective, which power is ample for diagnostic purposes. The thick-shelled ovum with its rugged exterior is an easily recognised object. Its characters I will not detail in this paper. They can easily be found in any of the works on tropical diseases. And I have yet to meet with the case where, having failed to find them in the stools, vermifuges caused the expulsion of the worms. I can, however, readily conceive that if there was only one small worm in the intestine, and the patient was constipated, the ova might be missed. In this case, however, I doubt if any symptoms of disease could be referred to the presence of the parasite. Barring the microscope for a time, the symptoms and signs I have detailed above should lead one without difficulty to the diagnosis of the affection. And it is always to be borne in mind in tropical countries that obscure gastro-intestinal symptoms ought to make one suspicious of the presence of intestinal parasites.

III.—Treatment.

Granted that the practitioner has formed his diagnosis, how should the disease be treated? Fortunately the answer is simple: *Give santonin.* In this drug we possess an agent which is practically a sure and certain power when administered rightly. But given in a haphazard and careless fashion it may not only be useless, but may produce most alarming symptoms of its own. Its administration should be varied to suit the particular case. Roughly speaking, all cases fall under two classes, those with urgent symptoms and those without urgency. In the former case nothing answers better than a dose of ol. ricini ʒi., santonin gr. iv., stat. It must not be thought that a single dose of this is enough. After the lapse of a week it is better to give one of the prescriptions advised below in the treatment of chronic cases. In giving the above dose the patient must be enjoined not to vomit, and should he (or she) do so, immediately after taking it, it should be at once repeated.

In the chronic cases I find nothing answers so well as:—Santonin gr. i.; hyd. c. cret., gr. i.; M. vi., 1 bis die. If thought advisable this can be given continuously for several weeks, without any ill effects. It will be noticed that I have chosen hydrarg. cret. in preference to calomel. I have used the latter drug and am quite aware that it holds a position of some repute as an anthelmintic. My own experience, however, has not proved to my satisfaction that, in the case of round worms at least, its administration is a great advantage, as compared with hydrarg. c. cret., while its drepressing effects are very much greater than those of this drug.

A prescription I have found of great use is as follows: Santonin, gr. i.; hyd. c. cret., gr. i.; quin. sulph., gr. iii.; VI., 1 bis die. It is not uncommon out here for the patient whose worms are being disturbed to be attacked by a sharp attack of fever. In some cases I believe this to be due to the presence of the worms, and to the absorption from the intestinal canal of some toxin of whose nature I am ignorant. In other cases the fever is undoubtedly malarial, and I have found the plasmodium in the blood. In the former class of cases I have failed to find anything abnormal in the blood. Where a patient is constipated and the symptoms are not urgent, it is often a great advantage to clear the lower bowel thoroughly before commencing the santonin treatment. For this purpose I use calomel and jalap or colocynth and hyoscyamus in the form of a pill.

In prescribing santonin the patient should always be warned about the probability of zanthopsia, the colouration of the urine and the possibility of an attack of fever, such as I have described, being set up. This is an extremely important point, especially among a people like the Chinese.

Another rarer result of the administration of santonin is a sharp attack of vomiting. This, however, passes away without treatment in twelve to fourteen hours. I have never seen any case of cerebral disturbance caused by santonin, but such cases have been reported. My readers will notice that I have not discussed the utility of any other vermifuge. This is of deliberate intent. I am quite satisfied with santonin properly administered, and have not found any drug to match it in efficiency. And as the purpose of this paper is practical, I do not wish to cloud the main points by side issues.

In conclusion, let me urge on such of my readers as intend to work in the East the extreme usefulness of a practical knowledge of the intestinal parasites and their ova.

A CASE OF BLACKWATER FEVER IN TRINIDAD.

By R. C. Bennett, M.B.C.M.
Government Medical Officer.

Blackwater Fever is so extremely rare in this Colony that the record of a case may serve a useful purpose. In fact it was believed not to exist in Trinidad, and there were grounds for this belief as no cases were reported. During the last eighteen years' practice I have heard of only three cases, and the case under review is the only one that I have seen and treated. It is worthy of note that all these cases occurred recently (two this year and two in 1898-9) and in different parts of the Island. Is hæmoglobinuric fever a recent introduction into Trinidad, or have cases been overlooked or returned as "Malaria," with all the appendages so often affixed to it?

Case: L. R., aged 36, white subject and Government officer, has resided for many years in outlying and unhealthy sub-district, where he was seen July 28. He then had slight fever (with rigor) and vomiting which came on at 3 p.m., on his return from his ordinary duties. He had been having these attacks of fever regularly for two or three months, got rather accustomed to them and treated them lightly, though his general health was being affected. He had taken quinine at irregular intervals. There were no means of examining the blood on the 28th, patient being seventeen miles from my residence. Within a few hours after my departure he forwarded sample of urine which he described as "black as ink," "voided with difficulty and slight pain," and added, "I am yellow all over, my eyes especially so." Without delay I had him brought out, forty-two hours after visit of 28th. This comparatively short period had practically wrecked him, and he was in a critical condition for next forty-eight hours.

There was marked yellow discoloration of skin, and sclerotics were especially so. This had come on suddenly. Spleen was enlarged, not to any extent; liver not. No diarrhœa. Constipation and nausea. Temperature 100-5°, weak, general prostration, urine deep dark port wine colour (almost black), scanty (not sufficient to obtain specific gravity), neutral (or alkaline). Albumen, a sample coagulated in tube. Amorphous *débris*, some hyaline casts, no blood corpuscles.

The urine separated into two distinct layers on being allowed to stand. Twenty four hours after it was crowded with bacterium (bacillus) ureæ.

Patient's blood was carefully examined, but no malarial or other parasites could be found (he had been taking much quinine). Many of the red cells were crenated and buckled.

Within a week the patient was transferred convalescent to a town residence of his family. The urine then was clear, free of albumen, and the yellow discoloration of skin had disappeared. He was, however, anæmic, weak, emaciated, and extremely debilitated.

Treatment.

On general principles, with stimulants.

Remarks.

(1) The sudden onset of urgent dangerous hæmoglobinuric symptoms in a case of ordinary chronic intermittent malarial fever, in an area in which the disease had never been heard of before invites attention.

(2) It has been stated that blackwater fever is caused by a parasite like that causing Texas fever (pyrosoma bigeminum).

(3) No parasites were found in this case.

(4) What was the cause of the blackwater fever in this patient? How explain the sudden transition of an ordinary intermittent malarial fever into an almost fatal blackwater fever in an area where blackwater fever was neither epidemic nor endemic?

(5) Professor Koch would say quinine caused it. Admitting that quinine in large doses "exercises a certain amount of destructive action on the blood corpuscles rendering their hæmoglobin unstable" (Manson), practical experience renders Koch's theory untenable. It would be more in keeping with the maximum amount of reasonable possibility to consider blackwater fever a "post malarial condition"—occurring in cases that have been saturated with malaria—whose resisting powers have been gradually lowered, and in whom an abnormal amount of malarial toxine has been accumulated, and suddenly exploding as it were into acute hæmoglobinuric symptoms (rapid destruction of red blood corpuscles and loss of albumen).

I have never heard or read of a case of blackwater fever that had not previously been the subject of ordinary malarial fever, and malarial fevers are due or now well-known malarial parasites.

An Epidemic amongst Rats and Pigs is reported from Nicaragua. During the month of August of this year hundreds of these animals were found dying or dead in the streets and elsewhere. The suggestion that the disease is plague is being enquired into.—*Medical Record*, September 1, 1900.

CIRCUMSCRIBED CUTANEOUS ŒDEMA.

By A. B. Dalgetty, C.M., M.D.
South Sylhet, India.

Dr. Pounett's article with the above title, in the Journal for July, leads me to record a somewhat similar affection which is occasionally seen amongst tea-garden coolies, and which has always been a puzzle to me.

The patient comes up to the dispensary some morning complaining of stiffness and swelling of one hand, which has come on during the night. On examining the part, the dorsum of the hand and wrist will be found to be considerably swollen and to pit slightly on pressure; but beyond the inconvenience and stiffness caused by the œdema, there are no other symptoms. I have not noticed any constitutional symptoms.

The patient cannot account for the condition. The bite of a centipede or the sting of a bee will cause a similar swelling, but the sufferer is always aware of it, if such has been the case. Besides, the œdema from these causes subsides in a few hours, while that of the disease under discussion takes as many days.

I have seen at least six cases, five of which were women, and in all the part affected was the dorsum of the hand or the lower part of the forearm, but I have never seen both hands attacked at the same time, nor observed it on any other part of the body.

My cases were not noticeably anæmic, nor had they been suffering from malarial fever, or from any constitutional affection. In two cases it has recurred months afterwards.

The time within which the œdema disappears varies from two days to about twenty days, some stiffness of one or more fingers being the last to leave.

The only treatment I have tried is ungt. iodi; it seems to aid absorption. Sometimes the coolie ties a string fairly tightly round the wrist above the upper border of the swelling, with the idea that it prevents the œdema spreading upwards.

Thinking that the local œdema might signify the presence of filaria or some other blood parasite, I took great trouble with one case and examined numerous fresh blood films, both during daylight and after dark, but I did not succeed in finding anything. The ætiology is a complete puzzle, and the text-books do not mention the disease.

Leprosy at the Cape.—Leprosy would appear to have been introduced to the Cape by European colonists, although none of the immigrants came from leprous countries. At present no class or race, whether Hottentots, Kaffirs, Half-castes, Malays or Dutch, are free of leprosy, but it is after all a rare disease in South Africa. In the *Polyclinic* for October the principal dates as to the appearance of leprosy in South Africa are given. Three cases were noted at Drakenstein in 1756. Graaf Reinet had a leper asylum in 1817. About 1817 leprosy increased amongst Hottentots and Bastards, at which date a leper asylum was instituted at Hemel en Aarde, and more recently at Lovedale, near King William's Town.

Recent Experiments in Malaria.—In the last (September) issue of the Journal, we announced the fact that Dr. Manson's experiment of importing malarial-fed mosquitos from the Roman Campagna to London, and there allowing the insects to bite healthly persons, resulted in establishing his views on the matter. The malarial parasite was found in the blood of two "volunteers" who submitted themselves for experiment. Mr. P. Thurburn Manson was the first to develop fever, and afterwards Warren, a laboratory assistant at the London School of Tropical Medicine, became ill. As the disease was of the benign tertian type, neither volunteer was inconvenienced for more than a few days, a few doses of quinine serving to rid them of the parasites in their blood. This experiment, combined with the non-infection of Drs. Low and Sambon, who took up their residence in the Roman Campagna during the summer of 1900 in a mosquito-protected hut, will surely convince the most sceptical that the mosquito is the one factor in malarial infection which has to be dreaded.

The Second Session of the London School of Tropical Medicine was opened by Sir William MacGregor, K.C.M.G., on October 3. The distinguished company which assembled to listen to the opening address assembled in one of the new wards recently added to the Seamen's Hospital Society at the Royal Albert Dock. The address will be found elsewhere in the Journal, and we hope in our next issue to describe the new wards, which both from a practical and æsthetic point of view are unsurpassed by those in any hospital we have seen either at home or abroad. The school continues to flourish, and the number of students indicates all too plainly the necessity for increased accommodation.

Plague.—The plague in *Glasgow* appears to be disappearing, at any rate for the time being. In all there have been twenty-eight cases of plague, twenty-five of which have been admitted to the Plague Hospital; six of the hospital cases died, and three deaths occurred in the city before the hospital was opened for plague cases.

A death from plague occurred on October 4, at Cardiff in Wales; the patient arrived at Newcastle from Rosario on the River Plate, and although feeling ill travelled by train to Cardiff, where he died. This is the second case brought to our shores from South America, the first patient travelling *via* Cardiff to Hamburg, where the case was diagnosed. In *San Francisco* plague would seem to have been endemic in China town since March of this year. In *India* the recrudescence of plague which threatened in the month of August seems to have abated. In *Poona* and at *Belgaum* the numbers of plague deaths during September increased considerably, but neither outbreak has been attended by a serious mortality. In *Hongkong*, *Mauritius* and in *Queensland*, *Australia*, a few cases of plague continue to occur in a more or less sporadic fashion. Judging by the cases which come to British shores, the greatest danger at present would seem to be from South America. By report the outbreaks in Rosario, Asuncion and Santos would seem limited, but they would appear none the less dangerous to the rest of the world.

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THE

Journal of Tropical Medicine

OCTOBER, 1900.

ARCHITECTURAL REQUIREMENTS FOR TROPICAL DWELLINGS.

THE architect who may hope for success in designing dwelling houses in warm countries must be prepared to conform to the dictates of science. It is now proved beyond doubt that the bite of the malarially infected anopheles claviger is the chief (it may be the only) means by which malaria is conveyed to man. This has been proved not only conversely but obversely as well, for granted a protection from mosquitoes the disease cannot be contracted. Drs. Low and Sambon have kept good health whilst dwelling for months in a mosquito-proof house, in what is termed the most unhealthy district of the Roman Campagna. Unhealthiness applied to almost any part of the world generally implies malaria, and it now appears that the ill-fame of any locality is traceable to the bite of the mosquito. Remove this cause and the unhygienic region is converted, if not at once into a salubrious health resort, at least into a spot where fair health may be possible. The small hut in the Roman Campagna in which Drs. Low and Sambon have lived during the summer of 1900 will be a historical abode; for it is a prototype of what every tropical dwelling must in future be, if "fever" is to be defied. The principles of its construction will remain for all time, let the materials of which the house is composed be what they may. Every human dwelling and, if the greatest good is to be done, every cattle shed or byre, must be mosquito-protected. It seems incredible, it almost appears faddy, to insist that our tropical dwellings should be built so as to be proof against what seems an insignificant insect. The pioneers, in the movement to have their houses built mosquito-proof, will have to bear the brunt of being considered faddists, but they will have the laugh in the long run, when health proves to be theirs.

There is no architectural revolution needed; it merely requires that air on entering the dwelling shall be filtered of mosquitoes, and luckily the means of effecting this is neither expensive nor harassing. A copper-wire gauze over windows and doors, with meshes fine enough to prevent mosquitoes passing through, is all that is required to render a house "healthy." It may be objected that a wire gauze over every window would darken the rooms by excluding light, and would render the house "stuffy" by keeping out a free circulation of air. But it must be remembered that the mosquito window-guard need not be in place during the day; it should be drawn one hour before sunset and not be withdrawn until one hour after sun-rise. If, however, this is inconvenient, the windows in the house should be multiplied; the unprotected windows, which admit light only, need never be opened, and the protected windows, which serve to let in air and keep out mosquitoes need never be closed. In a house thus built, every second window would be mosquito-proof, alternating with light admitting windows. By the windows thus permanently

guarded other pests besides mosquitoes would be prevented admission. There are several tropical pests besides mosquitoes, although none are known to be so dangerous. Flies are believed to be carriers of the poison of several diseases, such as typhoid, plague, &c.; centipedes, tarantulas, white ants, flying beetles and several other insects which cause annoyance, and in some instances danger, to tropical dwellers, would be effectively excluded as well. Even snakes would find it difficult to gain entrance and it is plain that by the protection of doors and windows the insect worries of life in the tropics would be reduced to a minimum.

Messrs. Humphreys & Co., of London, have produced the first mosquito-proof house, and we would recommend them to seriously consider the architectural steps necessary for more permanent dwellings. The needs are few, but the good to be gained is incalculable and the devisers of a fever-proof abode will deserve well of almost all mankind.

A HOLIDAY IN THE MOSQUITO-PROOF HOUSE IN THE ROMAN CAMPAGNA.

By D. C. Rees, M.R.C.S., L.R.C.P.
Superintendent of the London School of Tropical Medicine.

The editor of the Journal of Tropical Medicine suggested to me that an account of my visit to the mosquito-proof house in the Roman Campagna might prove interesting to his readers. I conceded to his request with the greater readiness because some of our former students and others have written to me from time to time, asking if I could tell them how the experiment was progressing. Well, I can assure them that it is a complete success. This has been a bad season in the Campagna, and practically everyone, excepting Drs. Sambon and Low, have suffered from malaria.

I arrived in Rome on September 8, and after spending one short day at sight-seeing I turned my back on the "Eternal City" and made my way to the Campagna. It proved to be a hot and dusty three hours' drive to Ostia, the place where the little English colony has made its temporary home. On my way I could not help observing how deserted and forsaken the country side looked. One felt intuitively that some influence was at work which had mastered man, and as I came across the inhabitants and saw their sallow faces, their listless and languid air, it became evident that that influence was malaria. The people were all cachectic, most of them in the advanced stages of it.

On arriving at the mosquito house a very different scene presented itself. Drs. Sambon and Low came running out to meet me, looking the picture of health and vigour. I received a very hearty welcome from the two exiles, and was at once made one of their party—on certain conditions, however, namely, that I promised to be indoors every night before sundown, and to abstain from taking quinine. To these conditions I readily assented. The house is comfortable, and consists of five rooms, one of which serves as a laboratory and mess-room combined. The windows are protected with copper wire gauze, having a mesh of about 1 mm. It is fitted with a double door, which is similarly protected. The door was locked every night at sunset, and no one was allowed to enter after that hour. The house is situated in probably the most malarious spot in the whole Campagna.

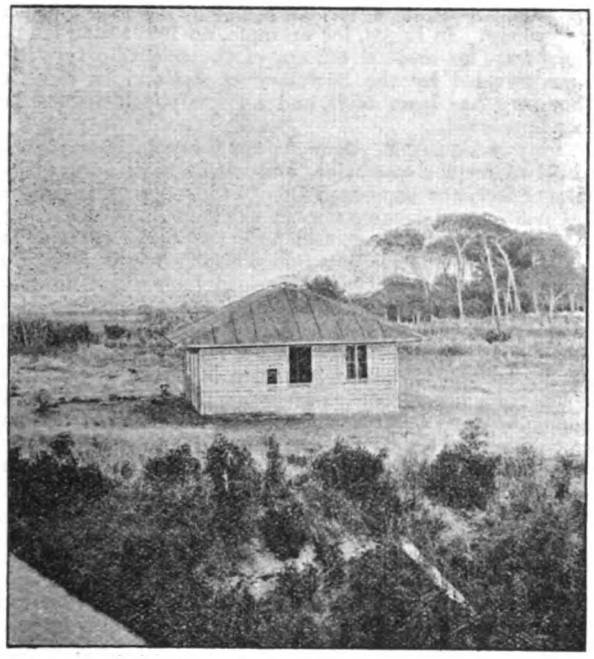
Mosquito-proof Hut in the Roman Campagna.

It is practically on the banks of one of the main canals, which at this season of the year is literally swarming with Anopheles claviger. Near at hand there is an outhouse which contains Dr. Low's heterogeneous collection of animals, birds and reptiles; namely, snakes, frogs, lizards, pigeons, bats, mosquitos and filariated dogs. This family was presided over by a pet owl, who exercised his royal prerogative from time to time by killing some of the most valuable members of the collection. Dr. Sambon, however, would not think of parting with it. He said the household would be incomplete unless presided over by an owl, the emblem of wisdom.

The days passed quickly and there was always plenty to occupy our time. The animals had to be looked after and their blood examined. Blood films were sent in by the neighbouring doctors for diagnosis. Mosquitos were collected and dissected, the various pools and canals were explored for mosquito larvæ.

Villages were visited and outbreaks of fever in men and cattle investigated. Visitors had also to be received and entertained. A large party came one day consisting of Professor Grassi, Drs. Foa, Noé, Zupitza (Hamburg School of Tropical Medicine) and others. On another day A. Plehn came down to see us. Professor Grassi was very favourably impressed by the way in which the experiment was being conducted, and at the end of his visit despatched a highly congratulatory telegram to Dr. Manson. I do not know if this has been published.

We always turned indoors before six o'clock, and we used to stand at the windows and time the first appearance of the Anopheles, they would come as regularly as clockwork; they seem to scent their prey a considerable distance away. It must have been very tantalising for them to be unable to get at us.



Mosquito-proof Hut Showing Surroundings.

I should like to mention here, that so far as the Campagna is concerned, the Anopheles does not bite during the day. I can safely say that I was never bitten, although we were out and about the whole day. Of course when collecting mosquitos and going to their haunts, one instinctively keeps an eye on them. When sitting smoking a pipe in the evening, with not a single mosquito or insect pest to annoy one, I could not help thinking how extraordinary it was that mosquito-proof houses had not come into use years ago. We used always to sleep with our windows wide open, so that if marsh air had anything to do with malaria we should have contracted it.

I was in the Campagna when the rains set in and everyone said, "Now is your time, you will all get fever." Two of us purposely went out in the rain and got soaked to the skin, but we disappointed our neighbours, for we got no fever. On going into Ostia in two or three days' time, however, we found practically every one down with it. They crawled out of their houses looking the picture of woe and misery, and gave us their hot feverish hands to feel. The explanation of this is of course evident, the inhabitants are all malariated, and chilling caused by the rain determined an explosion of fever.

My visit came to an end all too soon, and I left my two hosts with a feeling of great indebtedness for the trouble they had taken to make my holiday so enjoyable as well as profitable.

I have purposely avoided any reference to the scientific work that is being done; an account of this will appear in due course in the report that is to be published.

I shall look forward, also, to some graphic pictures of the life of the peasants of the Campagna, from the pen of Dr. Sambon. The lot of these poor people is a hard one—that of the African savage is enviable in comparison to theirs.

AN ADDRESS ON SOME PROBLEMS OF TROPICAL MEDICINE.[1]

By Sir William Macgregor, K.C.M.G., C.B., M.D., D.Sc., LL.D., &c.

When in compliance with the request of Dr. Manson, consented to deliver to-day the opening address to the students of this School of Tropical Medicine, I felt I was committing myself to a task really beyond my powers. This feeling was deepened on glancing over the formidable syllabus of lectures and demonstrations to be given by the teaching staff. The ground to be covered is so vast, the subjects to be dealt with so very important, the extensive and diversified knowledge required, both in teacher and student, is so deep and wide, as to make one's own ignorance painfully apparent to oneself. I therefore find comfort in the fact that I am here to-night because I am an administrator, not because I am a doctor of medicine.

I understand that the objects of this school are to convey to its students a special acquaintance with the diseases that are of most frequent occurrence in tropical regions, and to train them for the investigation of such maladies on systematic and scientific lines.

There is excellent reason for believing that Her Majesty's Secretary of State for the Colonies took very seriously to heart the deplorable mortality among public officers and others in our more unhealthy tropical possessions. The calling into being of this school is but one of the many means devised or fostered by the Right Honourable Joseph Chamberlain for curtailing the death roll of our fellow citizens in those insalubrious, over-sea territories of the empire. This school is but one link in the chain, and the chain is already a long one, and promises to be continuous.

The Training of Colonial Medical Officers.

The leading idea of the school, therefore, is the appropriate training of colonial medical officers. It is, however, to be hoped that others may take advantage of it, that it may add another ring to the ever-widening stem of the already gigantic tree of medical knowledge. It is certain that there are a great many people in this country at the present moment suffering from what are usually called tropical diseases, and that these sufferers are not, and cannot be, attended by medical men that have had tropical experience. Very many of these patients must come under treatment by

[1] Delivered at the London School of Tropical Medicine, on October 9, 1900.

the ordinary medical practitioner. Cases could be adduced in which some of the most distinguished men in the medical profession in this country have been completely at sea as to the exact nature of certain tropical diseases and their sequelæ. The teaching supplied by such a school as this should be a useful addition to medical men practising in this country, especially if cases of tropical diseases in sufficient numbers can be made available to them for clinical study. The list of students that have passed through the courses since October 1, 1899, does not show, however, that the school is patronised by the British medical practitioner. Taking into account the already immense and still growing traffic, the rapid and steadily accelerated communication between this country and the tropics, and bearing also in mind the intolerance of any form of quarantine in the United Kingdom, the probabilities are that the special study of tropical diseases will constantly be more and more forced on medical men in practice in this country, either by the systematic study of such maladies at the ordinary schools of medicine or at specific institutions, such as this is. If this branch of medicine is neglected, then the sufferer from the tropics will be a certain loser, but not always the only one.

At the present moment, however, many of us are very specially interested in this school in relation to the medical services of our more unhealthy tropical colonies.

It is probably the case that not a few of those that have so far passed through this institution were already familiar with many tropical diseases, knew them by experience, and understood how to treat them. To students of that category the clinical study of these maladies here is not nearly so important as is the training to be had to fit them to investigate tropical diseases on the spot, on the most advanced systems known to medical science, and with a full knowledge of what has already been done in these matters in this country and elsewhere.

Any average medical officer that has sufficient training in making investigations of this kind will soon master the practical details of all tropical diseases in his district, provided he is given time, the necessary instruments, and proper accommodation. The best and most proper place for making such researches is undoubtedly the tropics themselves. It must be patent to every person that thinks at all on the subject that the training of medical officers to carry out investigations of this kind is of the utmost importance to each colony, to the empire, and to humanity at large. We all know, or ought to know, what has been done in this field by such men as my late highly esteemed friend Dr. Bancroft of Brisbane; by the distinguished Dr. Manson, the inspiring genius of this school; by the father of Strachan's disease, the present chief medical officer of Lagos; and by many others that have practised medicine in the tropics. These men had no specialised opportunities given them; they had not the advantage of any systematic training such as is offered now at this school. We are therefore justified in expecting a great deal from the younger men that now come forward here to learn at once the most suitable and most advanced methods of conducting original investigations in the great and rich tropical field.

State Encouragement of Medical Research.

But I can tell you that this training and preparation will not yield all that we are justified in expecting from it without the co-operation of the Secretary of State and of Colonial administrators. The trained medical officer must have time and opportunity granted him. That the Secretary of State will in every possible way encourage, and suitably recognise, the researches of medical officers may safely be taken as granted. It is, however, at times a very difficult matter for an administrator so to arrange service affairs that he is able to grant to medical officers the time so necessary to carry on scientific investigations of this kind without their being submitted to grave or destructive interruptions. Colonial administrators and others in high authority have not always in the past lent to the head of the medical department the support required by him. They have not always given to their chief medical officer the confidence that Alexander the Great extended to his. I was myself very fortunate in the first new country in which I served in having as chiefs such enlightened men as Lord Stanmore and Sir William des Voeux. Whatever was really required for the new medical service in Fiji was granted. As one result of that there is no doubt that at the present moment the chief medical officer of that colony, Dr. Glanville Corney, is thoroughly familiar with the practical aspect of every disease met with in that country. Many other Colonies have been less fortunate, but if that is so now, Mr. Chamberlain, who not only thoroughly understands the value of medical work, but also takes the deepest interest in it, and has done more than any other person ever did before to popularise scientific medicine, is just the man to see that medical matters are put right wherever they are wrong; they are, in my humble opinion, wrong where original work cannot be done. My own personal experience has been that the most unhealthy Colonies are, or were, those in which medical research was most neglected; that, however, is apparently being remedied everywhere. In Lagos, for example, no fewer than ten full equipments for medical officers of the scientific type have been provided by the Secretary of State. An excellent laboratory has been built, and an analytical chemist has been appointed.

This has been done there without any reference to any school of tropical medicine, and would have been carried out precisely the same had there been no such institution and no medical commission in existence. We look, however, to this school now, as a means of better enabling our medical officers to take advantage of the opportunities that will henceforth be given to them in Lagos, and no doubt similarly in other Colonies, for you may depend on it that Colonial administrators will know their duty sufficiently well to follow with zeal the course that has been taken by Mr. Chamberlain. Moreover, there is at the present time in this and other countries a volume and force of public opinion behind sanitary and medical work that has, perhaps, been dormant from the days of Moses and Machaon till now. It was only the other day that the celebrated Grassi lectured on malaria before the Queen of one of the Great Powers of Europe. Such an event is redolent of the times.

It is not my intention to occupy your time in speaking of the details of your training for the investigation of disease, beyond emphasising their very great importance. I would rather in a familiar way give those of you that have not yet practised in the tropics some idea of the extremely interesting and very important work that awaits you there.

Old Diseases and New Races.

You will still, for example, have many opportunities, especially in new countries, of acquiring valuable information respecting the original or natural location of disease, the landmarks of which are fast disappearing before modern civilisation. You will see how old diseases are being communicated to new people, and be able to watch the strange results produced. You will doubtless be in a position to add much to the existing knowledge of tropical diseases already more or less studied, and you will in all probability be able to establish the existence of maladies at present unknown and unrecognised; you will thus reduce the sum of human suffering. Can any man desire greater glory?

As different forms of disease were localised originally, and still remain more or less so, it follows that the experience of tropical medical officers differs largely according to country. Their opportunities are therefore diversified in an unusual degree. Perhaps I may personally have had exceptionally good opportunities of assisting—as a French writer observed some time ago—at the dissemination of disease among new races.

Typhoid Fever.

The late Sir Thomas Watson was very anxious to know

Fig. 1.—The caudal extremity of *Culex* ♀, the light brown mosquito of China.

Fig. 2.—The caudal extremity of *Culex* ♂, the light brown mosquito of China.

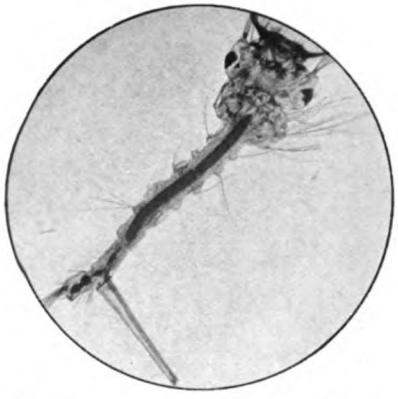


Fig. 5.—The first moult stage of *Culex* larva from Bombay, showing great length of caudal tube, and large size of cephalic portion.

Fig. 6.—The caudal extremity of mature *Culex* larva.

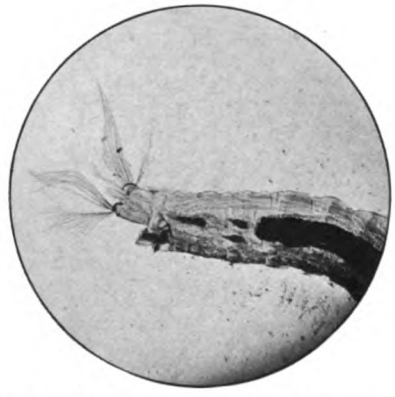


Fig. 7.—The caudal extremity of the *Anopheles* larva (well grown) from Bombay.

Fig. 3.—The caudal extremity of *Culex* ♂, the small tiger mosquito of China.

Fig. 4.—The caudal extremity of *Chironomus plumosus* ♂, water midge of England.

OBSERVATIONS OF MOSQUITOES.

By P. W. BASSETT-SMITH, Staff Surgeon, R.N., F.Z.S.

Bale and Danielsson, Ltd., London.

whether typhoid fever was known in Polynesia. From inquiries made there in 1875 it appeared certain that this disease did not occur in the islands. In 1875, however, a case was observed in a gentleman recently arrived from Australia. It assumed the virulent form that is accompanied by petechial spots, and the patient died. Soon after this a case occurred in a European in the mountains of Viti Levu. The disease, it appeared, had been contracted in the locality where the first case was treated. Sporadic cases were met with at intervals, but they were, formerly at least, few in number. Perhaps their scarcity was largely due to the fact that houses were supplied with rain water collected from the roof and stored in metal tanks. There was, however, a form of continued fever in Fiji that very accurately simulated typhoid fever up to the ninth day, when it suddenly culminated, and the patient got well in a very few days. Typhoid fever was also unknown in British New Guinea. In that country the continued fever that in Fiji so closely resembled typhoid was not met with. Typhoid fever is also absent in West Africa. It would appear to be a by-product of civilisation, which is perhaps also true of cancer, diptheritis, and some other diseases.

Fiji, by the mere fact of becoming occupied by a considerable number of Europeans, was a centre for the distribution of other diseases to her neighbours; but she sometimes received their maladies in return, as will be seen from what follows. The disease of yaws has been from time immemorial endemic in Polynesia. If a Fijian child escapes yaws terrible results are expected to the future health of a being so abnormally constituted. Yaws represented our measles, scarlet fever, chicken-pox, diptheritis, and whooping-cough in one.

Yaws.

The probability is that no medical officer will be long in the tropics before he is brought into contact with yaws. This disease, like most others in the torrid zone, is of course parasitic. I first met with it in the Seychelles Islands, in African slave children liberated there by our cruisers and indentured as plantation labourers. It is curious that this disease is much more mild in the African and Papuan than it is in the Polynesian. It may be that it is newer to the latter. In the African it is sometimes so ill-marked that only a medical practitioner of some experience can recognise it at first sight. It is not very contagious to Europeans, but when caught it is loathsome and troublesome. I have never looked on yaws as a disease that could be cured under from three to six months.

I was once invited by my superiors to take up the extermination of this disease in New Guinea, but being then the only medical man in a colony larger than Great Britain, and having absolutely no means of dealing with an endemic contagious malady, I was obliged to say, in somewhat different words, that I might as well try to arrest an earthquake. In Polynesia, if not in New Guinea, there is not only the endemic nature of the disease to contend against, but also the very strong native prejudice of "the conscientious objector" in its favour, a prejudice difficult to deal with, though the disease is not made an object of worship there as is the case with smallpox in West Africa.

Though yaws was indigenous to Fiji, tinea imbricata was not known in that colony till introduced by plantation labourers. Many Line Islanders were brought to Fiji as plantation hands, and among them tinea imbricata is endemic, while yaws was unknown to them. The Fijian gave yaws to the Line Islander, and the latter gave his loathsome tinea to the Fijian. The Solomon Island recruit also brought tinea imbricata to Fiji with him, and he in turn left the colony, in a great many cases, with venereal disease in some form or other.

Venereal Disease.

Venereal disease was not known to the Pacific Islanders, nor to the Papuans, till taken to them by Europeans. Certain social habits of these peoples facilitated the rapid spread of these maladies, and they flourished with the usual luxuriance of a crop on new soil. It was very extraordinary how the Pacific Islander was affected by soft chancre. Very frequently the sore developed into a great spongy mass that bled profusely if touched. In not a few cases the ulceration caused death by penetrating the abdominal wall. These specific sores were also remarkably contagious. On the other hand, syphilis, being more slow in its processes, did not immediately effect the ravages that might have been expected. Gonorrhœa was all but incurable in native women. At first many of them were brought to Fiji as plantation hands. It was extremely rare that one returned home with a child. The trader had some small share in spreading these diseases, but it was insignificant compared to what was done by the labour traffic. Yet in the face of all this, some good Churchmen, and at least one Bishop, that visited the recruiting islands expressed themselves as favourable to the labour traffic. These good men did not see behind the calico, "*Come storpiato è Maometto.*"

Measles.

Fiji was also the centre for the distribution of measles in 1874, a disease introduced there by one of Her Majesty's ships of war. The epidemic that followed destroyed between a third and a fourth of the whole population, affording a striking example of the exuberance of a new disease among a new people. In this respect, however, measles was not more surprising in Fiji than was the usually insignificant disease of itch in British New Guinea. This latter malady, introduced in some old clothes, sped like a tidal wave along the coast, and in a few weeks thousands of people were covered from head to foot by the most exaggerated form of the disease. It probably spread much faster from the fact that one shirt might be worn by several people in one day. When matters were beginning to assume a somewhat serious aspect, the disease, having spent itself, began to abate, and soon declined to the level it will probably always keep in a community where clothes are common property and itch attacks the human face.

Dysentery.

There are few scourges that require the same thorough and exhaustive study that dysentery calls for. This disease, in its epidemic and contagious forms, was unknown in British New Guinea half a score of years ago. In recent years it has caused many deaths there. It began in Fiji with the advent of a European population, and led to considerable mortality among them, even before annexation. But during the prevalence of measles, and frequently afterwards, it assumed an epidemic form. From the first introduction of the disease there it was regarded as contagious. When settlement became better established, and the houses of Europeans were provided with rain water stored in metal tanks, dysentery among them became much rarer. But on large plantations, and in native villages, it took on at times a very malignant form. Instances occurred in which even 50 and 75 per cent. of the Polynesian labourers of an estate died of this disease, which was new to them. Excessively high mortality from the same cause occurred also on some of the Queensland plantations among the Polynesians employed there. I was once received at a certain plantation by an interesting-looking boy of about 14 years of age, who was evidently not of the usual class of recruits. On being asked who he was, he said he was So-and-so from New Britain. To the question, "How many are there of you?" he replied, "Plenty all die, only me." I found there had been one hundred and fifteen of them, and that he was the sole survivor. This was the work of dysentery. The treatment in each epidemic was at first like groping in the dark. In a very deadly outbreak that occurred amongst some three score of recruits landed out of a vessel in which they had been battened down for thirty hours during a hurricane, an examination of the contents of the intestines of those dead but an hour or two showed that these consisted of a mass

of what was then called vibrios. In consequence of this observation a mixture of corrosive sublimate was given to the two score survivors, all in hospital. The remedy acted like a charm and not another case ended fatally. But this remedy failed utterly in two or three succeeding outbreaks elsewhere in the colony. During one season salicylic acid acted with extraordinary success. Then it in turn ceased to be of any use.

From many observations of this kind it appeared that dysentery is made up of entirely different diseases, or of one disease that presents widely diverse stages. It offers a splendid field to the humanitarian and to the enthusiastic scientist that we trust may be sent out from this school. Were I to judge from my own personal experience I should say dysentery causes more deaths than any other disease in tropical countries. No other malady is so universally distributed and of such constant occurrence.

Laveran says "Le paludisme est certainement la plus répandue des maladies endémiques," and Manson says of malaria "It is the great disease of the tropics." My experience is different. I have served in two countries where there was no malaria: never in one where people did not die of dysentery. Last year of 39 cases of dysentery treated in Lagos Hospital, 10 died, say 1 in 4; of 128 cases of malarial fever, 4 died, 1 in 32. Dysentery is the chief agent in the rapid depopulation of the Pacific. It swells the death rate in West Africa, but not so much amongst Europeans as amongst natives. But there one does not see the wholesale depopulation from this disease that is sometimes so clearly observed on a Pacific island from the return home of a single labourer suffering from contagious dysentery. There can hardly be any doubt as to the contagious nature of some epidemics of dysentery. We regarded the common house fly as an active agent in its spread in plantation hospitals, and took precautions accordingly. The communicability of this disease has always to be taken into account in the tropics, more particularly in places like West Africa, where the sewage of a number of houses often flows into the great clay pit from which the houses have been built, and out of which the same houses draw their water.

The man that will work out an effective and practical means of dealing with contagious dysentery will be the greatest benefactor of the races that live in the tropics. He may claim to be the saviour of the Pacific Islander, the most lovable man of men now living. It is a study that I most earnestly commend to your attention and on which I trust you will all turn the searchlight of science. Dysentery is a destructive giant, compared to which strong drink is a mere phantom.

Malaria.

To the tropical European, though perhaps not to the tropical native, the most important study is probably that of malarial fever, the investigation of which has already furnished us with some of the finest examples of human intelligence, perseverance, and observation, and unveiled to us some of the most wonderful workings of Nature. To myself this chain of marvels, full of poetry and religion, nowhere better seen than in the splendid illustrations of Drs. Ross and Fielding-Ould, always recall the words of the second greatest Teuton of the century:—

> Wie alles sich zum Ganzen webt,
> Eins in dem andern wirkt und lebt!
> Wie Himmels Kraefte auf und nieder steigen,
> Und sich die goldenen eimer reichen!
> Mit segen duftenden Schwingen,
> Vom Himmel durch die Erde dringen,
> Harmonisch all das All durchklingen!

The prediction that malarial fever would be found to be due to a parasite having its cycle in man and the mosquito, is fit to be compared with Goodrick's theory of the cycle of Algol; with Leverrier's and Adams's assigned position of Neptune; with Murchison's prediction of gold in Australia. To my own mind it recalls the early teaching of the germ theory of antiseptics by the immortal Lister. It may not improbably lead to an equally great revolution in medicine. The position assigned by foreign writers to our British scientists in this: the greatest of recent discoveries, is and must remain most honourable to them and to our nation. The name of Dr. Manson, one of the brilliant lights of this institution, will always be connected with the fertile hypothesis that has led to the astounding revelations of that prince of observers, the great Ross, of Grassi, Celli, Bignami, Bastianelli, Koch, and others.

The whole subject of malarial fever is of more importance to the Italians and to ourselves than to any other nation. The Italians have, they say, eleven millions of themselves exposed to it, which, according to Celli, furnishes them with two million cases a year, with an average mortality of fifteen thousand. It is true that it is almost unknown in Great Britain itself. But Laveran states: "Morehead estimit qu'aux Indes les fièvres palustres comptaient pour 40 sur 100 dans la mortalité générale." This is probably not proved. Manson at the Royal Colonial Institute showed what a serious matter malaria is on the West Coast of Africa. This is enough to demonstrate of what vast national importance it is to the British Empire.

There seems to be some tendency at the present time to seek the origin of malarial fever in coloured tropical children. Surely it must be acknowledged to be an old domesticated disease in the United Kingdom and on the Continent of Europe, for which aboriginal children could not at any time have been held responsible. You all remember how in the beginning of this century the Anopheles completely routed one of the most powerful British armies ever sent to the Continent of Europe. Then, again, our history supplies few more pathetically ridiculous pictures than the kingly founder of the Royal Society dodging his doctors to take his Peruvian bark on the sly. Of course Dr. Schwalbe would say at once that the King contracted his fever abroad. As the two chief attacks occurred nineteen and twenty years after His Majesty "came to his own," the probabilities are strong that he owed them to the genuine domestic English Anopheles.

In these modern investigations into malaria the Italians have nobly done their share; so have the Germans, through the illustrious Koch. We owe, further, a very great debt to Laveran. I have often been asked by non-medical friends who has done this great work, whether it is all British? It appears to me to be more or less like this: Manson was the surveyor, Laveran made the road, Ross built the bridges and laid the rails, and Grassi, Bastianelli, Bignami, and Celli provided the rolling stock.

Grassi says: "Preciso perciò che la scoperta che gli anofeli inoculano la malaria umana è uscita dal mio cervello, seguendo una via da me ideata. Naturalmente anche la mia scoperta, come moltissime altre, non sta isolata, ed io non ho mai esitato a dichiarare che sono parvenuto ad essa giovandomi della ipotesi dei mosquitos svolta tra gli altri da Laveran, Manson," &c.

Angelo Celli writes: "Sotto i consigli del Manson, cioè del celebre parasitologo che aveva gia descritta la vita della filaria nel corpo delle zanzare, il Ross, maggiore medico inglese nelle Indie, fece pungere da zanzare uccelli. . . . (e) ricostrusse le fasi del ciclo di vita nella zanzara. He speaks again of 'questo che possiamo chiamare ciclo del Ross.'"

M. Emile Bertaux thus expresses himself: "Les deux observations initiales sur lesquelles repose la nouvelle théorie de la malaria n'ont pas été faites en Italie. C'est au docteur Laveran qui revient l'honneur incontesté d'avoir observé, dès 1880, le parasite dont la présence dans le sang humain est la cause directe et unique de l'infection palustre. Mais c'est le médecin Anglais Ross, qui., le premier, determina rigoureusement l'agent de transmission d'une maladie analogue à la malaria humaine."

Koch tells that: "Ueber das eigentliche Wesen der Malaria haben wir erst in der neuesten Zeit Erlklaerung erhalten durch Laveran."

As long ago as 1892 Laveran used these prophetic words: "J'ai émis l'hypothèse que les moustiques jouaient un rôle dans la propagation du paludisme comme dans celle de la filariose." You know how powerfully Manson championed that doctrine, based as it was on his own original and independent work.

A great deal still remains to be done in connection with this subject, perhaps much more in tracing analogies in other diseases than in connection with malaria itself. There are now many workers in the field, and doubtless their ranks will be strengthened from this school.

The Destruction of the Mosquito.

To my mind the parasitic cycle in malarial fever is proved by demonstration. Although this is so, the experiments now being carried out by some of our countrymen here and in Italy are by no means superfluous. Those that care to read Dr. Christy's book will find at pages 27, 29, and 69, that similar experiments have already been made by the Italians, by whom they have been fully described. But it is highly desirable that they should be performed in a more sensational form, and in British blood, to impress and convince the British public. These experiments are the more to be commended that they can be carried out without any greater risks to the subjects of them than is incurred by any European that lives for a few days on the West Coast of Africa, perhaps beyond reach of doctor, nurse, or any other European. The chief use of these experiments is to demonstrate the truth of the theory advanced. There is, of course, no comparison between the position of men sent into a malarial region provided with every appropriate appliance, and with the sole duty of protecting themselves from mosquitos, and the case of those that mount guard, that nurse the sick, that tend machinery at night; or to the case of the man belated in "the bush," or stranded on a mud flat.

Even in its present state of development the new doctrine of malarial fever is such that no conscientious administrator could take the responsibility of ignoring it.

In the West African coast it must now be reckoned with at every step. Hospital management must be fundamentally affected by it. Wards will have to be painted of a colour that will facilitate the discovery of mosquitos; and probably some of them will have to be furnished for fever patients with doors and windows of gauze wire netting Every bed, without distinction, will have to be provided with a fine muslin mosquito net. All water tanks must be supplied with wire net coverings to prevent the ingress of mosquitos. Wells and reservoirs and flower pots will have to be similarly protected. In hospital discipline it will be considered a serious offence to allow a fever patient to be bitten by a mosquito. All mosquito breeding places near a hospital or other dwelling will have to be made unfit for these insects, as far as this is practicable. Much attention will have to be given to the teaching of the new doctrine. All hospital nurses must obtain a mastery of the subject; and so, of course, must sanitary inspectors, otherwise they will be unfit for their posts. But the general public also must have the leading lines of malaria genesis put before them in a way they can understand. It should form a subject of tuition in all the public schools of a place like Lagos, and prizes should be given to the best scholars in the malaria class. The nervous individual that does not know one genus of mosquito from another will, in future, lead an unenviable life in the tropics. Ladies that understand the mosquito theory will not dine in low evening dresses; nor will gentlemen sup with their ankles under the table and covered only by black thin silk socks.

The steps mentioned above, and many others like them, are all very obvious, and seem very simple in theory. I regret that I cannot completely share the rosy optimism of our leaders and teachers in this matter. I do not overlook the fact that malaria has been practically extinguished in this country, which is not congenial to it for reasons of meteorology. Malaria in the tropics is much more difficult to deal with than typhoid, rabies, or small-pox here, and those are not yet vanquished. It is to be feared that in a country like Lagos, when all that can be done shall have been accomplished there, the results may be somewhat disappointing. Lagos is on a lagoon that crosses the territory from east to west, at some places four or five miles broad. It is all fresh water except near the town of Lagos, where it is brackish. We know from the Italians, and by our own experience, on the great western coast of British New Guinea, where there is no fresh water, that the Anopheles can breed in brackish water. The lagoon water is full of aquatic plants; it has very little current; it rises and falls probably five or six feet; it cannot be drained; it cannot be enclosed within banks; it cannot be kept at the same level. One thing could be done; Lagos Island could be surrounded by a sea wall that would render the current there generally too fast for the Anopheles, according to the measurements of Celli. It is believed in Italy that the velocity of water, to ensure against the breeding of the Anopheles, must not be under about 1,900 yards an hour. Then, Lagos Island contains very likely 200 acres of swamp, with all sorts and sizes of water puddles. These could be filled up. If, however, the statement of Grassi is well founded that "gli Anopheles si sviluppano la dove sono . . . . paludelli anche microscopici," mosquito hunting at Lagos will never lead to the extermination of the Anopheles there.

It may be said that the remedy for all this is to abandon Lagos town, and go further inland. Alas! we cannot transport the lagoon, and it is necessary for our commerce. A considerable number of people must remain at Lagos, even if the seat of Government is shifted to some other place. My own opinion is that were the harbour opened, the island surrounded by a sea wall, and the swamps filled in, Lagos would then be fairly well protected from fever, but not otherwise.

In the absence of these costly undertakings we must trust more to the paradox of preserving the Anopheles from infection than to anything else. We must give quinine to all and sundry gratuitously, and we must see, especially, that it is given to children. Our fever patients and our convalescent stations must be carefully guarded, and kept, when possible, where there are no mosquitos. Our medical staff is numerically weak. We must train native youths in Lagos itself to the extent of making them able to deal with such things as small-pox, malaria and dysentery. This has been done elsewhere, and therefore should be carried out at Lagos.

If the sanitation of the Lagos railway now under construction is not thoroughly taken in hand on sound principles two results will follow: the railway will increase malaria; increased malaria will augment the cost of working, perhaps make the railway a financial failure. We have many swamps in our large towns, and huge clay holes in all towns and villages to deal with; add to this that our population is half naked. We have also to face the immense fecundity of the mosquito. Ficalbi says one mother mosquito may in the fifth generation be the progenitor of twenty milliards. Howard shows that one rain barrel may contain 19,110 larvæ, and that they may produce at least twelve generations in one summer. This at seventy eggs a mosquito would produce in a summer a number of mosquitos expressed by twenty-five figures. These are among our difficulties. In our favour we shall have the support and encouragement of the Colonial Office; a chief medical officer that is himself a successful worker at the malarial theory; and we shall have a number of medical officers trained here. It is also in our favour that we have no rice fields; that our soil is sandy and dries very quickly, resting on a shingly subsoil. It will be interesting to watch the result of a malarial campaign under such circumstances.

A very important point to determine at Lagos is the distance an Anopheles will fly to a feeding ground. Will

they cross the lagoon? To Mr. Fagan I am indebted for the recent work on mosquitos by the American, Howard, referred to above. From it we gather that mosquitos may travel fifteen miles on a light wind. Celli, whose new book cannot be spoken of too highly, admits that mosquitos will extend, probably three miles in a horizontal direction. I have had painful verification of that on the New Guinea Coast and on Lagos lagoon. Celli seems to cite the case of Sezze to show that the limit of oblique propagation of the mosquito is attained at an altitude of 1,000 feet. Koch finds them at 3,000 feet in fever communities in Java. We were punctured by mosquitos in our camp on Mount Scratchley at 10,000 feet. They were very troublesome at 5,000 to 6,000 feet. Mere altitude cannot therefore be taken as a safe guide to safety from fever. Before we can shift establishments to the Olokemeji hill, 1,000 feet high, and fourscore miles from Lagos, we must station residents for a year on the hill, in order to ascertain how far it would be fever-free.

My own personal experience of the Anopheles has been somewhat interesting. I never saw one in Fiji, where malarial fever is, or was in my day, unknown. Dr. Finucane tells me it is still absent there. But amongst the recent consignments of mosquitos from Fiji, Mr. Theobald has found the remains of one Anopheles. If it is established that there are Anopheles mosquitoes there, the matter becomes one of much scientific interest. Fiji would therefore be in the position of Mauritius and Bourbon before the terrific outburst of fever there in 1867; that is to say, there would be given a country hitherto free of malaria, but furnished with Indian coolies with fever blood; from which at any time the propagation of malarial fever may be set a going in Fiji. So far Mr. Theobald has not had any Anopheles from Mauritius. No doubt they will turn up soon. I first saw the Anopheles on the afternoon of the first day we visited the west coast of British New Guinea, about nine years ago. I saw something on the forehead of Mr. Cameron, one of my officers, that looked like a small brown peg, and was surprised to find it was a mosquito standing as it were on the end of its proboscis, projecting nearly at a right angle to Mr. Cameron's forehead. It soon filled with blood and began to void it till a drop of blood fell from it to the ground, on which Mr. Cameron thought he had enough of it. We all had sufficient experience of it before night, for it is not the case that the Anopheles bites only at night, nor that its puncture is always painless. On the contrary, we found it often as sharp as a prick from a needle. Of course all mosquitos love dark ways, and will prefer to bite at dusk; but the Anopheles will not hesitate to feed, at all events on a dull day or among trees, when it gets the chance. Specimens were then sent to Mr. Savile Kent, who pronounced them a new mosquito.

It seemed strange that we were repeatedly camped for weeks at a time in the mud and swamps of the western or Anopheles country, and yet left without any cases of fever. The reason is that for some hundred and fifty miles of coast there were no human inhabitants, which would seem to show that blood diet is not necessary to the hatching of Anopheles. We had been at work a couple of years in the central and eastern districts and had suffered much from fever there before we visited the west, yet the Anopheles was new to me when I saw it there. Koch has, however, shown that the fever parasite is very common in Kaiser Wilhelmsland, from which it may be inferred that I overlooked the Anopheles in Eastern New Guinea. Those that suffered most from fever in British New Guinea were the crew of the steam yacht *Merrie England*, who were far more frequently ill than those people that travelled daily in swamp and forest, no doubt because Anopheles had become domesticated in the men's quarters on board.

Now we require to know how and why mental excitement, any considerable change of temperature upwards or downwards, whether caused by the sun, cold draughts, rain, or other agency, should bring on an access of fever long after possible infection and in spite of long continued and large doses of quinine. In my own person an access of fever seems to be sometimes caused by exposure to bright sunlight, without reference to temperature.

Texas Fever.

In British New Guinea we never doubted that the horse and dog suffered from malaria, but this part of the problem seems to be solved by the experiments of Koch, which show that even the fever parasites of man and of the anthropoid apes are different and not transferable from one to the other.

Although the specific nature of the human parasite seems thus proved, it still leaves open for investigation on the West Coast of Africa a cognate question of great social and economic importance, to which I venture to invite your special attention—namely, that of the disease that makes it so difficult to keep a horse alive in the forest country near the coast. I do not scruple to suggest that you should investigate this equine malady or maladies, for two reasons. In the first place, it is of very great importance; and, in the second place, if medical men do not examine it, it is not probable that any other competent person will do so. I for one shall be surprised if it is not found that the disease is a parasitic one, though poison has been often suspected. Could I find the money, I should be prepared to advise the Secretary of State to offer a handsome prize to whomsoever should discover the means of rendering horses immune to this fatal disease. My fellow-administrators are enterprising men, and something may yet be done in this direction by combination.

Koch[1] is convinced that a practical inoculation against Texas fever can be arrived at. This and his observations on the Surrakrankheit,[2] coupled with the observations of Smith and Kilborne, are sufficient to justify any such expenditure as that proposed, showing as they do that certain breeds of animals are already immune to the tsetse malady and to Texas fever.

Elephantiasis.

It has been announced lately that the mosquito has been found to be concerned with that strange disease, elephantiasis. This is a malady that I have seen in man on perhaps every part of the body, from the crown of the head to the sole of the foot; but I have never witnessed it in any other animal.

In practice and in origin there does not, according to my experience, seem to be any connection between elephantiasis and malarial fever. In Fiji, where there was no malarial fever, elephantiasis was very common, in certain small islands phenomenally so. The largest scrotal tumour removed there weighed 122 lbs. There were several over 80 lbs. in weight. In British New Guinea, where malarial fever was very common, elephantiasis was rare. The same is true of Lagos. It would therefore seem that these diseases are in those places more or less in inverse relation to each other. I have not heard of any case in Lagos nor in British New Guinea where a European contracted elephantiasis. Two European officers, at least, became affected by it on the Island of Rotumah. Though, therefore, it is a matter of minor importance compared to dysentery or malarial fever, you will still find elephantiasis to be a very interesting subject for investigation.

Phthisis.

It does not appear that phthisis was a disease known to the Papuan. The natives of New Guinea are, however, very subject to a form of pleuro-pneumonia common in epidemic form at the beginning of the cold season, apparently contagious and most likely parasitic, to which many succumb. Phthisis was not originally a Fijian disease. Its primal distribution you will find to be a question to the solution of which you can still contribute something.

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[1] *Aerztliche Beobachtungen in den Tropen.*
[2] *Reise Berichte*, 1898.

Diphtheria.

Neither in Fiji nor in British New Guinea was there any such disease as croup or diphtheria; they had not reached the latter colony when I left it in 1898. The first case of diphtheria that occurred in Fiji was about twenty years ago, in a little girl 7 or 8 years of age. This little patient brought the disease from Sydney, and both the child and her mother died of it. A few weeks later a case appeared among the prisoners in gaol in the same town. Sporadic cases occurred later, and probably the disease has now become domesticated in that country, but it is, I believe, still rarely met with there. It does not appear to occur in the Lagos territory in West Africa, and Koch notices its absence in German East Africa.

Cancer.

In some of these new countries there are interesting points to note with regard to cancer. I do not remember to have ever operated on a Polynesian or Melanesian for cancer, though I had to do so several times on Europeans in Fiji. For nine and a-half years I never saw a case of cancer in British New Guinea, but at the end of that time there occurred an example of encephaloid cancer of the tibia in the person of a Papuan that had for seven or eight years lived practically a European life, eating tinned Australian meat daily. It seemed hardly possible that he could have become infected from any previous case. Lupus exedens is very common in new Guinea, but it is always clearly distinct from cancer. The latter disease is of course very prevalent in Australia. It may be remarked in this connection that the Polynesian and the Papuan are practically vegetable feeders. Dr. Johnson of Lagos, now in this city, tells me that in Lagos during a practice of fourteen years' duration, he has five times seen cancer in native patients, and that in each case the sufferer had lived as Europeans live. In West Africa there are, however, very marked cases of destructive lupus.

Tetanus.

An equally remarkable experience was met with as regards tetanus. In the disturbances that took place in Fiji in 1876 and 1877, when the mountain chiefs refused to recognise the authority of the Queen, and Lord Stanmore had to reduce them to obedience by force of arms and without any army, we treated about one hundred and fifty wounded men, suffering from all kinds of injury. Although the nursing and dressing must have been very imperfect, and operations were performed under difficulty, there was not a single case of tetanus.

On the other hand, an officer that received very serious injuries from the explosion of a charge of dynamite in the interior of British New Guinea, died of true traumatic tetanus. He was camped at the upper end of the alluvial country on a piece of ground cleared by us, close to the foot of the hills, some 60 miles from the sea. Four or five days later, after a journey down the river, during which we camped each night on its alluvial bank, he was got on board the steamer, and a few days thereafter showed the unmistakable symptoms of tetanus. He was taken straight to the Cooktown Hospital, and died there of this disease. No case of tetanus in man or animal had ever been heard of before in British New Guinea.

It is not clear that the case of Commodore Goodenough, reported to have died of tetanus from an arrow wound received at Vanikoro, is parallel to this one. It is quite possible that the detachable bone point used by Papuans and some Micronesians on the tips of their arrows may in his case have been left in the wound and have caused septic poisoning. These bone points, prepared by scraping down a piece of human bone, would contain septic matter, yet I have not known these to produce tenanus in New Guinea. It may here be mentioned that erysipelas was unknown in those countries.

Tinea Imbricata.

The steady healthy progress made by a new invading disease was well seen in British New Guinea in the case of tinea imbricata. It was a domesticated disease in the West, on the Fly River, for example, where it is known by the same name that is applied to tobacco. It was also domesticated in the east end, where it is called by a word that in a certain district means "to curl" or "fold up." The central district was still free from it as recently as ten or twelve years ago, but it is slowly and surely gaining on that region, and during the last three or four years cases have been appearing even at Port Moresby. In a few years it will be endemic over the entire coast, a great eastern wave from the Line and Solomon Islands meeting a wave equally great and irresistible coming from the west. There, as in Fiji, the best treatment was found to be sulphur fumigation, but it is not possible to apply the remedy universally in New Guinea so as to extinguish the disease.

Ankylostomiasis.

I can only mention by name several other tropical diseases I have met with that are of special interest and that require investigation. There can be no doubt that ankylostomiasis is indigenous in the Pacific. In 1876 it was found in Fijian mountaineers, and in recruits from the Solomon Islands. Perhaps some of you may be able some day to explain its wide distribution and its exact significance. Cerebro-spinal meningitis was not a native disease, but appeared amongst Indian coolies in Fiji. How it came there was a mystery. Beri-beri was not, I believe, a Fijian malady, but has been introduced by plantation labourers from Japan. In New Guinea it is indigenous but not common.

Unknown Diseases.

In New Guinea there is a curious multiple tumour often met with as large as a walnut. It occurs most frequently about the elbows or the parts of the body that touch the ground when one is asleep on it. This has not been investigated. It is probably parasitic. In the Pacific there is a strange *post-mortem* appearance that is extremely common, and that, I trust, some of you may throw light on. In the hilum of the kidney there is very often half a teaspoonful of brown or purulent fluid. It is found in the greater number of bodies, even if dead only an hour or two. Its cause or origin, so far as I know, has not been made out.

Dingoes and Rabies.

As an example of the kind of medical legislation as to which you will have to give advice, I may first cite from my own experience with rabies. As you all know there is a species of dog, the so-called "dingo," native to Australia and New Guinea. Where that animal came from is not clear. My distinguished friend, Professor Giglioli, of Florence, gives good reasons for regarding the Australian aborigines as degenerated Aryans. If they are so, they probably brought their dogs with them, and at a time when rabies did not exist in Aryan communities. We shall not consider here whether the barking of dogs is a modern accomplishment, or whether the dingo gradually forgot the art. The dingo and dog cross readily, and the first cross between them barks. Doubtless, therefore, the dingo would be readily susceptible to rabies. There is a wild dingo on the top of the Owen Stanley range in British New Guinea, and a complete specimen was secured and deposited by me in the Queensland Museum, but the result of its examination is not yet known to me. The domestic dingo is very common in New Guinea as a pet, as a hunter, and as a table delicacy. All the dingoes of a village meet several times a day to howl, and they never separate without a fight. They are provided with long, thin, sharp teeth, which they use in season and out of season, but by preference when out of sight. It closely resembles, or is identical with the Australian dingo. I have never seen a native dingo in the Pacific Islands.

The point that most concerns us here is that the Australian and New Guinea dingoes were free from rabies. The Australian colonies have protected themselves from rabies by instituting a term of quarantine on imported dogs. It was seen that rabies introduced into a country like New Guinea

with swarms of ill-tempered biting dingoes, among a barefooted, bare-legged population, and with a wild dingo in the mountains and numberless pigs everywhere, would be a very serious matter not only for that country but also for the whole of Australasia. The matter was therefore dealt with on the broadest platform. A representation was made to the great colonies that had charged themselves with part of the burden of maintaining British New Guinea, urging that the Secretary of State should try to induce France, Germany, and Holland to introduce laws similar to those of British New Guinea, to prevent the introduction of rabies into their possessions in those seas. The then Prime Minister of Queensland, Sir Samuel Griffith, saw at once the importance of the measure and warmly supported it; it was, I believe, also approved by the Government of New South Wales. The then head of the Government of Victoria thought the proposal more or less ridiculous, and was of opinion that France and Germany would give no attention to such a trifle. Lord Knutsford and Lord Salisbury, however, obtained at once the cordial co-operation of the three foreign Governments. There is therefore good reason for hoping that now the whole of Australasia is, with reasonable care, secured for all time against the terrible disease of hydrophobia.

The "Jigger."

The question of the introduction of the "jigger" is one that has recently occupied the attention of the Indian Government. To such matters even as the control and distribution of this flea you require to devote time and study. Guided by our experience in Lagos we could only report to the Secretary of State that the "jigger" does not cause any noticable inconvenience on official expeditions, and appears in private life to claim very little attention. But a medical officer advising his Government in India, would require to know a great deal about the insect before he could venture to recommend any legislative steps regarding it, or could suggest that no action whatever should be taken.

Leprosy.

It is probably now generally conceded that the disease of leprosy is communicable, and that the isolation of lepers should be carried out in the interests of the community. No aboriginal people I have known have ever entertained the slightest doubt as to the contagious nature of the disease. The natives of West Africa isolate the lepers. In the South Seas they kept them apart, and often buried them alive when they reached an advanced stage of the disease. More than twenty years ago I read in a German author that leprosy was extinguished in Scotland "durch die Entmannung der Männer." I have not been able to find the authority for that statement, the author of which is dead many years. It was at one time apparently not uncommon in Scotland. In the *L'Histoire Universelle* of M. Rambaud it is stated that Scotland's most celebrated king died of this dread disease.

The whole subject of leprosy now requires to be studied exhaustively in the light of the new pathology.

No doubt some of you must be aware that the native Fijian treatment was to bake the sufferer from leprosy near a slow fire. It was confidently affirmed that cases were cured in that way. It will be very interesting to see whether a fuller knowledge of the disease will offer any explanation of such a cure.

Quarantine.

I would for a moment call your attention to a medico-legal subject of great importance to many Colonies, and to which I trust some passing attention may be given in the courses of this school. I allude to quarantine against the introduction of infectious or contagious disease.

In all international conferences on quarantine matters British delegates take their stand against the imposition of quarantine. Their system is the isolation of affected or suspected individuals; the disinfection of vessels, and sometimes of cargo. This procedure can be carried out successfully only in a country where there are highly developed police, health, and sanitary services. It requires ample hospital accommodation where patients and prospective patients can be isolated; it needs a staff of trained and intelligent officers to exercise surveillance over people that may have been exposed to possible contagion, and it requires the means of dealing with any of these wherever he may be found. Here in the United Kingdom cleansing and disinfecting can be carried out with scientific accuracy and completeness, and at small cost.

In this country, then, in the face of the drainage, the ventilation, the accessibility to suspected persons, and the means of dealing with those as well as with infected persons, an outer line of defence may not at any time become necessary. But it is widely different in most colonies. Here the outer line of defence, imposing quarantine on infected or suspected vessels or persons, becomes a necessity. You will find it will tax your powers to the utmost to advise your Governments how best to combine the public safety with the minimum of disturbance to trade, for that in practice is the problem to solve. At Lagos we have *tried* to combine the two systems, by providing the medical department with the powers necessary to protect us from outside by imposing quarantine on arrivals, and to *exercise* surveillance ashore over persons exposed to a possible infection, but who cannot or need not be detained in quarantine. Ashore, the medical officers may remove from a community, and isolate an infected individual; or they may put a house, a village, or a district in quarantine. The small numeric strength of the staff available, the inadequacy of accommodation, the evil sanitary condition of all towns and villages over a great and populous area, make it practically impossible that we should depend entirely, or even chiefly, on our inner line of defence against epidemic disease. You are perhaps aware that in Australia a system of federal quarantine has been in force for several years. The geographic position of the Australian continent is very favourable to this line of action, yet recent events have shown that both the outer and inner ring of defence may be insufficient to keep out disease. The whole subject is one that will call for special and serious study at your hands.

The Water Question.

Let me impress upon you the extreme desirableness of becoming experts in the examination of water. Nothing is of greater importance in the tropics. Although the new doctrine of malarial fever shows that water is only an indirect agent in the production of that disease, it still remains an essential factor in its continuation and spread. It retains all its former importance in respect of other diseases. In West Africa the medical officer should be able to say whether a given well is safe or not on account, for example, of guinea worm. Water is popularly credited with the origin of the "craw-craw" disease in Europeans. It doubtless has much to do with the spread of dysentery, typhoid fever where that exists, cholera, and several other maladies. Altogether it is one of those subjects to which you cannot give too much attention. Personally, I have always contended that the quality of the water used by a community may have as much, or nearly as much, influence on them as has the quality of the food they eat, or of the air they breathe. A medical officer is constantly liable to be asked by his Government to report fully on the water supply of some place or community.

I cannot conclude these somewhat dry and disjointed remarks without offering my hearty congratulations to you that are to become students at this school. In my opinion your lot is the most enviable of any body of men in this great country. You have already travelled over the ordinary road of medical education and have become acquainted with the more frequented parts of the limitless field in which you are to labour. You have become ordinary members of the greatest, most beneficent, and therefore the noblest of all

professions. It can be said with greater truth to-day than it could three thousand years ago: *Ἰητρος γὰρ ἀνὴρ πολλῶν ἀντάξιος ἄνδρων.*

Here you are now to be taken into some of the bye-paths of medical study. There you will be brought face to face with some of the most interesting problems of science, questions of vast importance to the life and health of the individual, and that will exert a reflex action that will be felt in many of the transactions of public, national, and even of international life. I trust and hope that you will leave this school well fitted by your knowledge of your profession to do your part in the exercise of your calling, and that you will have securely planted in your mind that high sense of duty that is the life and soul of religion, of patriotism, of every efficient public service, and that enables the medical man to devote by instinct, and unselfishly, all his mind and all his strength to the prevention and cure of every disease that affects humanity.

British Medical Association.

THE HOT WEATHER DIARRHŒA OF INDIA.

By Major W. J. Buchanan, I.M.S.
Superintendent, Central Prison, Bhagalpur.

(*Continued from p.* 50.)

My object in writing this paper is to call attention to a very severe form of diarrhœa with watery stools, which is certainly not cholera, but in many cases somewhat resembles it. It is not difficult to understand that as hot weather in Europe is an important, if not the chief factor, in the production of "summer" diarrhœa, that the extreme heat of India should be *a priori* even more so.

Bowel complaints of all kinds are common in Bengal, but it is here intended to refer only to one particular form. It might be called thermic diarrhœa, and it has been called "choleraic diarrhœa." It bears, however, the same relation to cholera Asiatica as does English summer diarrhœa or "cholera nostras." Clinically, though it is very common in adults, it has a strong resemblance to what is called "cholera infantum."

Before going further it is well to give a short account of the clinical features of an acute case: The onset of the attack is always sudden, the patient having been apparently in his usual health up till a few hours before, then suddenly at night or in the early hours of the morning he is attacked with violent purging and vomiting; at first the stools are loose and fæculent, and the vomit contains the remains of the last meal; soon, however, the stools become abundant, more or less colourless, and pour out from the rectum as if from a tap. The patient becomes collapsed, with a shrunken cyanotic appearance, resembling, but in a somewhat less degree, that of cholera. The urine may be suppressed for several hours, the stools may number ten, twenty, or even fifty, in the first half-dozen hours. In old or feeble persons the result may even be rapidly fatal. More usually the violence of the attack grows less in the course of the day; a reaction sets in, which, however, is not so marked as is the reaction stage of cholera, the temperature may rise from 101° or 102° F., the stools become fæculent and only loose or semi-solid, and in uncomplicated cases the recovery is usually rapid. In a few instances the inflammation of the small intestine goes on to an ileocolitis and stools resembling dysentery persist for a few days. This is especially liable to occur if the patient has formerly suffered from dysentery. One might quote dozens of cases, but the following three will illustrate the different degrees of severity:—

(1) *Mild.*—J. S., aged 24, weight 107 lbs. On April 6, 1899, admitted to hospital at 5 a.m., with violent, loose, watery diarrhœa—ten to fifteen stools in the hour. Urine passed, no cramps, no collapse; voice good, recovery rapid, in the course of about six hours.

(2) *Severe.*—B. D., aged 38, came to hospital on March 23, at 5 a.m., passing thin, very watery stools with urine, vomiting frequent, partially collapsed and cold. When seen by me at 6 a.m. he was in this state; no urine for the past hour, but frequent scanty stools; very restless, face shrunken, voice clear, pulse rapid but perceptible; no cramps, tongue clean, urine remained suppressed for twenty-one hours. On second day the stools were semi-solid and next day he was practically convalescent.

(3) *Fatal.*—N. K., an old man, came to hospital at the same time as No. 2 above. Stools very watery, vomiting and nausea frequent; no urine passed, body cold, but pulse was just perceptible. In a few hours he was quite collapsed, passing frequent watery brown-coloured stools. Tongue soon became dry, the thirst was great. The stools never resembled "rice water." The diarrhœa continued, and he died at 4 p.m., after about twelve hours' illness. At the necropsy were found old pleural adhesions. The spleen was enlarged (8 by 5 by 2 ins.), no rice water stools in the intestine, but drab-coloured fæcal matter; one old patch of ulceration at the sigmoid flexure.

Albuminuria is often found in these cases, but does not last longer than a few days usually, where the kidneys have been previously sound. Constipation for a few days after recovery is common. Microscopic examination of the stools show abundance of epithelium, starch grains, particles of grain and husk, and numerous bacteria.

Treatment.—The treatment of these cases is not difficult. For attacks lasting only for a day food is not urgent, but boiled milk or milk and Mellin's food may be given. Brandy or rum will often be necessary. As regards drugs, it is unwise to interfere with Nature during the early stage of the diarrhœa, but by the time a patient is usually seen by a medical man many watery stools have been passed, so I usually at once give chlorodyne or camphoridyne (the tinct. chloroformi et morphinæ co. of the new *British Pharmacopœia*), sometimes with small doses of brandy, enough to control the diarrhœa. Then when the diarrhœa has become much less a dose of castor oil will set the patient aright. Care to be taken with the diet till the stools have become solid.

Ætiology.—As regards the ætiology of the disease this resolves itself into (1) bacterial and (2) predisposing causes. I have nothing certain to say about the bacterial cause. It is believed by many that the specific micro-organism of what Dr. Newsholme calls "epidemic diarrhœa" in England is the bacillus enteritidis, but as this micro-organism has already had attributed to it two diseases so entirely different as the well-known "rice pudding" epidemic of St. Bartholomew's (a sudden widespread and non-fatal diarrhœa), and the so-called colitis of English Asylums (a fatal disease which anyone with tropical experience would not hesitate to call dysentery), it is clear that one should hesitate to attribute such a widespread disease as summer diarrhœa to it also. We are on safer ground in discussing the predisposing, or rather the "exciting" causes. Granted the presence of the organisms of decomposition and of fermentation in the intestinal tract the actual exciting causes are as follows: (1) Badly cooked food; (2) hastily eaten food, or over-eating; (3) unripe or over-ripe food; (4) raw grain, eaten either carelessly or on purpose; (5) drinking too much water after or before food; (6) too rich food, curried and spiced; (7) badly prepared grain, especially maize, when weevil-eaten, as it usually becomes by the end of March in India; (8) over indulgence in alcohol, especially drinking freely at a big dinner by one usually abstemious, or a mixture of liquors—what is often called "upsetting the liver;" (9) excessive use of inferior fibrous vegetables, and many other similar causes. I have actually seen cases which were attributed to one or other of all the above causes. Some of them apply only to European habits and others to

the habits of natives of India. In gaols I am always on the look-out for bad cooking when such cases occur, for example, on one occasion in Midnapur Central Gaol I remember finding, one morning in May, no less than 125 such cases of violent diarrhœa admitted in one night; the cause was not far to seek. On the afternoon before a very violent cyclone had raged, with the result that the cooks were driven out of the cookhouse and the fires had to be put out, so that the evening meal of about one thousand prisoners was issued in a half cooked or rather half raw condition. On another occasion I had a series of 25 such cases admitted to hospital, owing, as I believed, to the temporary use of some bags of weevil-eaten maize, which were far worse than was expected. On this occasion it was remarkable that the cases only occurred from those on maize-flour diet, and not a single case among a large number of Bengali prisoners who got rice at all meals. Cases due to the use of over-ripe or half ripe fruit must have occurred in the experience of all who have lived in the tropics. One case I well remember, the patient confessed to having eaten the raw seeds of the jack-fruit tree, he suffered severely, having had suppression of urine for thirty-six hours and after the bowel complaint was over he suffered from successive crops of boils and his whole outer skin peeled off in handfuls, which symptoms I attributed to the endeavour made by the irritant poison to escape by the skin.

But while such factors as unripe fruit, raw grain, &c., are certainly important, I do not think that they are the whole cause; something more is necessary, and this something is the influence which is exerted by the hot weather on putrefaction or fermentation in the intestines. My reason for so concluding is as follows: I have seen all the above exciting causes in operation in cold seasons, but it is seldom or never that this violent form of diarrhœa is then produced; food may be badly cooked in the cold weather as in the hot, grain may be as badly husked, unripe fruit may be eaten, idle prisoners will eat raw grain in the cold weather as well as in the hot, but it is very rare for us to meet with these cases of diarrhœa in the cold weather. The same remarks apply to summer diarrhœa in England.

Diagnosis.—It is obvious that these cases in India must often need to be diagnosed from cholera. In cases where a bacteriological examination is not possible one must rely on the following considerations: (1) The presence or absence of cholera in the neighbourhood; (2) the low death-rate for the diarrhœa, and the usually high initial death-rate for cholera; (3) clinically by the fact that cramps are seldom present in diarrhœa, or if present, rarely severe, the collapse is not so complete as in cholera, the voice is never so low and weak as in cholera, the pulse though weak and small is never so low as in cholera, the stools are never the pure "rice water" of the graver malady, but always somewhat stained with fæcal matter. The reaction stage is less marked than in cholera cases, and some irritant cause is also usually to be found. I am strongly inclined to believe that in this form of "hot weather diarrhœa" the clue will be found to many of those mysterious cases of isolated sporadic attacks of "cholera." If "summer diarrhœa" can occasionally kill adults in Europe, I am certain that "hot weather diarrhœa" in India can do so.

Colonel Kenneth MacLeod remarked that this paper on hot weather diarrhœa raises two important questions; first as regards the distinction between cholera nostras and cholera Asiatica, and secondly, assuming that these are essentially different, or at any rate differently caused diseases, what the organism or toxin is which gives rise to the former? In the absence of bacteriological investigation the question of diagnosis is often very doubtful in India; but experience confirms the view taken by Major Buchanan in this paper, that these cases which are very common both in Europeans and natives, adults and infants, in the hot weather are not cases of cholera Asiatica. What the precise causation is we are ignorant, and it is best that we should acknowledge this, and content ourselves to await further research, which is urgently desirable.

NOTES ON A CASE OF BLACKWATER FEVER, WITH A DESCRIPTION OF THE MICROSCOPICAL APPEARANCES.

By George Thin, M.D.
London.

Dr. Douglas Gray, medical officer in the service of the Government of British Central Africa, wrote me from Zomba on March 27, 1899, advising me that he had sent me pieces of liver, spleen, kidney, and bone marrow from a very acute case of hæmoglobinuric fever, "our British Central Africa urinary suppression form." The case, he remarked, "was seen and studied by the Malarial Commission on the spot, and is referred to in their report;" and for this reason I should not have considered it necessary or useful to publish the results of my examinations of the tissues sent me by Dr. Gray, if I had not been asked to do so by Dr. Stephens, a member of the Commission.

When Dr. Stephens called on me after his return from British Central Africa, I offered to place my specimens at his disposal for any use he might wish to make of them, but he considered it would be more useful if I published a description of them myself; and writing me from Sierra Leone in December, 1899, whither he had gone on a second expedition, he remarked: "With regard to the specimens of the blackwater case, I think it would be of great value if you should describe them independently." This explanation is needed to explain how I come to take public notice of a case which is already in the hands of the Malarial Commission, and there is probably no more suitable medium for this supplementary addition to the account of the case than the Tropical Section of this meeting.

The following account of the case is an extract from Dr. Douglas Gray's letter to me:—

J. R., aged 27, marine engineer, been in British Central Africa eighteen months. On the whole enjoyed good health, occasional slight attacks of fever; rarely took quinine; strong, well-built man. On Sunday, the 19th inst., was walking about, but towards evening felt seedy. At 2 a.m. the following morning he suddenly vomited, and passed hæmoglobinuric urine two hours later. That day he passed about 8 ounces of urine, and never again, with the exception of 1 ounce about forty-eight hours later. He had violent hiccough and retching throughout, but very little vomit. He died on Thursday morning (the 23rd inst), at 5.25 a.m.—that is, three days and three hours from first prominent symptom.

Post-mortem examination revealed nothing special; liver, spleen and kidneys were all congested. Careful blood examination showed no malarial plasmodia. Intense anæmia (1,500,000). He lived ten yards from the bank of the Shire river. There were plenty of mosquitoes at some times of the year, few at others, notably at present; but they did not trouble him much, the bite and mark was very slight. He always used a mosquito net, and lived in the same house with two other healthy men. The previous death in the same locality was that of a gunboat captain, who also died of the suppressionary form of blackwater fever about ten months before. This case had certainly nothing to do with the abuse of quinine, as the patient very rarely took it.

The examination of sections made from the tissues showed the following appearances:—

Spleen.—White corpuscles containing malarial pigment in minute granules and very small spheres were found in the tissues of the spleen. They were comparatively most numerous in the Malpighian bodies, and were present in the large white corpuscles in the splenic pulp. They were also found in white corpuscles lying external to the walls of the small veins, and in the lumen of the veins; the number associated with the veins being larger than those found scattered sparsely through the pulp.

Liver.—In the liver white corpuscles containing pigment were found in the capillaries, and in small numbers in the

veins. Most of the pigment was found in the endothelium of the capillaries, the course of many of the capillaries being marked out by the pigment accumulated in the swollen and degenerated endothelial cells. Occasionally small particles of pigment were found between the capillary vessel and the contiguous liver cells. In many of the liver cells the pigment was found in minute granules in the substance of the cells.

Bone Marrow.—I did not find pigment in the sections of the bone marrow which I examined.

Kidney.—The sections of the kidney show the same degenerative and destructive effects on the secreting epithelium of the tubules that I found in a previous case from British Central Africa, which was sent me by Dr. Kerr Cross, and which I described in detail in the *British Medical Journal* of June 3, 1899, and as in that case there are great differences in the degree in which various tubules are affected, one in which the changes are in their extreme form being often found near a tubule in which the changes were scarcely observable. All the changes noticed are referable to the different stages of destruction of the epithelium.

The epithelial cells first become swollen and granular, then are detached from the basement membrane and block the tubule. The epithelial blocks gradually disintegrate until the final stage is reached, when the lumen of the tubule is found filled with a mass of minute rounded granules not unlike groups of large cocci. Every stage between the first and last change in the epithelial cell can be observed.

During the process of breaking up of the kidney epithelium the *débris* acquires the property of assuming different tints when stained with eosin. After the mass has become agglomerated, but before it breaks up, it shows, under the influence of this dye, a well-marked greenish tint, which still exists, but to a less extent, in the final stage of subdivision.

In the liver and spleen the dark colour of the pigment was accompanied with a distinct greenish-yellow tinge, and nowhere was it accumulated in large blocks. Its appearance and distribution in these organs was identical with that found in the tissues in the four fatal cases of acute pernicious malarial fever sent me from Sierra Leone by Captain Duggan, R.A.M.C., and in the previous blackwater case referred to.

The appearances in this case satisfy me that the patient died of acute malarial fever, in which the hæmoglobinuria was a concomitant symptom. The localisation and distribution of the pigment are evidences that it had only been recently thrown into the blood circulation. The absence of the parasite in the tissues of the spleen, liver, and kidneys does not militate against this view. The same absence held good in the previous cases to which I have referred, but in them the brain tissue was available for examination, and in it the parasites were found in great numbers. Had the brain in this case been available for examination, there is the analogy of the five previous cases to show that there is little doubt that it was in the cerebral vessels the parasites would have been found. The presence, condition, and extent of the pigment in the liver and spleen are sufficient to show the recent existence of the parasite in great numbers at some point of the blood circulation.

The changes in the epithelial cells of the kidneys illustrate the very powerful effect of a toxin generated by the parasite in these cases of blackwater fever. A reasonable explanation of the phenomena of hæmoglobinuria is that, in certain parts of the world, the parasite of malaria possesses the property of generating a toxin of such a virulent character that destruction of the red corpuscles, and the setting free of hæmoglobin in the blood, takes place on a large scale, while there is also a concomitant destructive effect on the epithelium of the kidney tubules.

Discussion.

I. Dr. Patrick Manson said it may be quite true that hæmoglobinuric fever is an expression of malarial infection (indeed, my own opinion, based, however, on considerations of probability rather than on logical deduction from demonstrated fact, is that it is), still I would protest against jumping at the conclusion that hæmoglobinuric fever is a form of malaria merely on the strength of the discovery of the malarial parasite in the hæmoglobinuric patient's blood, or of melanin in his viscera; concurrence of two facts by no means implies a cause and effect relationship of the two facts. Indeed, it would be strange if we did not find the *post-mortem* evidences of recent malarial infection in a large proportion of all cadavers of Europeans who die in such highly malarial countries as British Central Africa. It would not be justifiable to conclude because we found melanin in the liver or spleen in a case of cut throat in British Central Africa that the suicide was caused by malaria; neither is it justifiable to interpret its presence in hæmoglobinuric fever in a similar sense. Such rashness in accepting the concurrence of certain facts as being conclusive evidence is not likely to lead, I may say is likely to retard, the unravelling of the mystery of blackwater fever.

II. Mr. D. C. Rees said: We received the same tissue at the London School of Tropical Medicine from another source. I came to the conclusion that death was not due to an ordinary malarial infection. The amount of hæmoglobin in the kidneys and other organs was in such amount that if the case had been one of malaria, melanin would have been in much larger amount. I came to the conclusion that the melanin that was present was due to previous malarial infections. Other sets of tissues which we have examined lately tend to show that blackwater fever is not of ordinary malarial origin.

THE DISEASES OF GOORKHAS.

By Andrew Duncan, M.D., M.R.C.P.,

Physician to the Seamen's Hospital, Royal Albert Docks, London.

During the last year of my service in India, I had the good fortune to be associated with many Goorkha regiments; and as the result of this experience I found certain facts in their medical history to exist, which I had not previously noticed in connection with the men of other native regiments. These facts I propose to briefly put before you.

Malarial Fevers.—Shortly after taking over medical charge of the P.W.O.G.R.R., I was struck with the intractable character of the malarial fever in many cases as it occurred amongst the men. The fever proved to be peculiarly resistant to the usual anti-malarial remedies, so much so that in no other race in India have I experienced such difficulty in combating the disease. A number of drugs were tried, and I came to the conclusion that two methods of treatment only were of avail in these cases. First, the treatment by the rectal injection of 15 to 20 grs. of quinine. Should this not avail, then the only measure left was sick-leave to another district. Cases in which, for three or four weeks, 30 grs. of quinine had been taken by the mouth without avail, would often yield at once to 20 grs. by the rectum. Next to quinine, kreat gave the best success in these severe cases.

Phthisis.—The manner in which phthisis occurs, and its course, is also somewhat peculiar. Cases of an irregular fever used to come into hospital. Repeated examination of the lungs, which I was taught by experience never to omit, for many weeks would show no physical sign. Suddenly, one morning I would find sub-crepitant râles commencing at the apex. The early symptoms then almost invariably advanced with great rapidity. In fact, I have never seen such rapid cases of tuberculous disease of the lung as I have seen in Goorkhas. And in reference to this I may perhaps mention an extraordinary case I once had under my care. A Goorkha had been for three or four weeks in hospital when the signs of rapid disintegration of the lung appeared. Sub-crepitant râles were succeeded by crepitant râles, and a large mass of tuberculous glands appeared above the clavicle. So ill was he that for two or three days I went to the hospital

fully expecting he had died in the night. But one morning, to my astonishment, he appeared much better, and to cut a long story short, the signs abated, the gland enlargement disappeared, and eventually the man went out perfectly well. The treatment was by inhalation of creosote. I do not think there could be any doubt as to the accuracy of the diagnosis.

Mumps. — Goorkhas are very prone to mumps in their native country. Nearly always in the immediate few weeks subsequent to the joining of the new recruits, mumps would occur in the regiment. And the same remark must be made with regard to measles.

Ophthalmia.—Shortly after joining my regiment I was struck with the comparatively large number of cases of ophthalmia met with. I was at first inclined to suppose that the Goorkhas were in some peculiar way predisposed to this disease. However, I was called in one day to attend the wife of a Goorkha Rifle officer for phthisis, and was at once struck with the bad ventilation of her hut. Examination of the huts of the married and bachelor lines now showed the following important differences: Bachelor huts had no doors, had large airy windows opposite one another, fully 2 feet square, and kept open; the food was cooked in separate buildings. The married huts were irregular in shape, windows smaller, and not opposite one another, and generally half closed with articles of dress, and the food was cooked in the huts. The inner room was dark, and the atmosphere not fresh.

Next the incidence of ophthalmia was as follows:—

1895	... ...	Married, 30	...	Single, 11	cases.
1896	... ...	,, 68	...	,, 7	,,
1897 up to Aug. 28.		,, 50	...	,, 12	,,

On August 28 the regiment went on service and ophthalmia at once disappeared. I inquired of the medical officers of the other Goorkha regiments and found no such incidence of ophthalmia, with the exception of the 1st and 3rd G.R.R. Here the lines consisted of four airy double-storied barracks for a certain number of bachelors; the remainder of the latter and all the married men living in a series of tents closely aggregated under the brow of a hill. Twenty-one cases of ophthalmia occurred in 1896, of these 20 came from the latter barracks.

Enteric.—It has been often stated that the Goorkha soldier is prone to enteric. This has not been my experience. I have only seen one case of enteric (and this was judged to be enteric from the typical nature of the temperature chart chiefly) during my service in India amongst native troops. The case was, however, in a Goorkha. Surgeon-Major Armstrong, the Residency Surgeon in Nepaul, informed me that in his experience the Goorkha race is by no means prone to the disease. He has never had a case in hospital, civil or military in Nepaul, and has only seen two cases in females. So is that the fact!

A DISCUSSION ON ANKYLOSTOMIASIS.

I.—Major G. M. Giles, M.B., F.R.C.S., I.M.S., Sanitary Commissioner N.W. Provinces and Oudh, India.

The phenomenon of parasitism is so widely diffused throughout the animal kingdom that it is more than probable that the evolution of the parasite has commonly coincided with that of the host, so that it is by no means surprising to find Dr. Sandwith, of Cairo, quoting a translation of a Hieratic papyrus, 3,450 years old, which shows that even then ankylostomiasis was known as the A A A disease, though the writers assumed that the worms were the effect instead of the cause of the disease.

Quoting still from Dr. Sandwith's paper (written for the eleventh International Medical Congress held at Rome in 1894) we find that the earliest mention of this special anæmia in modern times was from Brazil, in 1648, and in Guiana in the beginning of the eighteenth century, but it was not till 1843 that Dubini, of Milan, first discovered the parasitic cause of the disease. After this Pruner (1847), Grasinger (1851), and Bilharz found it to be very common in Egypt, but these discoveries attracted no particular attention until the memorable outbreak of this disease amongst the workmen engaged on the construction of the St. Gothard tunnel brought its importance prominently to the notice of the medical world.

A few years later Leichtenstern and Lutz demonstrated the existence of a free stage, but owing possibly to climatic difficulties did not succeed in rearing the so-called rhabilites to sexual maturity; Lutz describing the still immature worms as becoming "encapsuled" and subsequently calcified. I believe myself that this is really a death and not a life history, and that the appearances described relate merely to the slow destruction of the embryos placed under conditions unsuitable to their well being.

Since the St. Gothard outbreak the disease has been recognised in all the warmer parts of the old and new world wherever civilisation and sanitation are so little developed as to admit of its progress. It has, however, not yet been certainly found above latitude 52° N., and above 47° N. the parasite apparently can only flourish in the sheltering warmth of mines.

This was then the state of our knowledge in 1889. As to the serious effects of the parasite on its human host all were fully agreed, for no one had then advanced the fantastic notion that it was a mere harmless tenant of the human bowel.

It was at this date the Indian Government became alarmed at the very high mortality prevailing in Assam, alike among the indigenous population, and among labourers brought from other parts of India to work in the extensive tea-gardens of that province. In the case of the latter, the causation of the mortality had already been made out to be ankylostomiasis by Dr. Ruddock, though it had unfortunately come to be known by the erroneous denomination of beriberi, while among the indigenous population the mortality was called kala-azar. I will not here enter into into the old controversy as to whether this increased mortality known by that name is due, as I maintain, to ankylostomiasis *plus* malaria, or to malaria *plus* ankylostomiasis, as advanced by Captain Rogers and Major Ross. We are all agreed that both these causes of cachexia are present in Assam, and it is probable that our real differences are of less dimensions than the volume would suggest. It is, however, obvious that, from my particular point of view, the exact ascertainment of the life history of the worm was the first importance. In the course of the investigation I was enabled to bring to light many entirely new points. Of these by far the most important was the demonstration of the fact that the full life history of ankylostoma duodenale is an example of dimorphism or heterogenesis.

My attention was first drawn to this by my finding sexually mature nematodes, the females heavy with eggs, which were absolutely identical in form and dimensions with those of the parasitic form, in ordure contained in a small pit such as is common in Assamese villages. I then set myself to see if I could breed a similar sexually mature free form in the laboratory, and after a few experiments hit upon the following method: a layer, about an inch in depth, of clean white river sand is spread over the bottom of a crystallising dish or other suitable vessel. Nematode worms are extremely rare in river sand, but it is well, before employing an apparatus so prepared, to moisten the sand and examine it for several successive days, to ensure that no such organisms are present.

Having selected a suitable case of ankylostomiasis—that is, a case in which repeated examination of the dejecta has demonstrated the absence of other parasites or their ova, such as the embryo of Anguillula stercoralis or the ova of oxijuris, &c., a small quantity of the dejecta is mixed with about three or four times its bulk of water, and poured on to the surface of the sand. The dish is then covered with a sheet of window glass, and placed on a shelf in the labo-

ratory, in ordinary diffused light. Every day a small portion of the thin upper fæcal layer is removed on a spatula, and mixed with water in a shallow glass tray and examined, either under the simple microscope or under a low power of the compound; but the former is preferable, and suffices for all but the study of the minute anatomy of the worms. Working in this way it will be found that the embryos hatch out in the course of a few days, the time varying with the air temperature from five days at a temperature of 60° F. to the next day at 84° F.; at 70° usually on the third or fourth day.

As found in the fresh dejecta the ova are usually 2-4 segmented, but unsegmented ova may often be found. Day by day it will be found that the specimens are more advanced, the segmentation gets finer, till a morula and finally a fully formed embryo is developed. It is useless to attempt to observe the further development of these embryos in clear water, for in this they cannot thrive, and slowly die for want of nourishment; but by taking samples from the cultivation at intervals and placing in water, the process can be followed with ease. On emerging from the egg the embryo measures 0·085 inches in length by 6·005 inches in diameter. At this stage they show little else than an intestine and œsophageal bulb. Within the next forty-eight hours (at 70° F.) they have increased to three times their original length and have undergone several ecdyses, the exact number of which I am unable to state, owing to the difficulty of keeping an individual probably nourished while under continuous observation, but roughly about twice in the twenty-four hours. In large fæcal masses under natural conditions, in the plains (temperature 80° to 90° F.), the embryos may develop into free sexually-mature worms in as little as four days, but under the somewhat less favourable conditions of my laboratory they occupied from six to ten days. As far as I could make out, the sexual organs were first clearly distinguishable in the last ecdysis but one, at which period the male measures about $\frac{1}{38}$ inches in length, by $\frac{1}{1000}$ inch. When completely grown up, he may reach $\frac{1}{24}$ inch in length by $\frac{1}{100}$ in diameter, while the females may attain $\frac{1}{15}$ inch in length by $\frac{1}{100}$ in thickness. It would occupy too much of your time to enter into the details of the anatomy and development of these free forms, for which I must refer you to my report on the subject, published by the Assam Secretariat Press, Shillong, 1890, but it may be well to meet here objections that have been advanced as to the validity of the above-mentioned observations, and as far as I have been able to follow the literature of these subjects, these objections are all based on a short paper by Dr. Sonsino (*L. Anchylostoma E. La Sua Profilassi*, 1889), the other criticisms I have met with being apparently mere mechanical transcripts of this paper, by commentators who obviously have not been at the pains to consult my report of the original. Dr. Sonsino's criticism throws doubt on the accuracy of my observations on the two following grounds:—

(*a*) That the organisms described by me are: Rhabdites terricola or other free nematodes; (*b*) that the appearances were really the free form of rhabdomena intestinale.

The first objection is obviously quite untenable in view of the method of examination adopted. Owing to the absence of suitable nourishment, clean river sand is, at best, an unlikely habitat for Rhabdites terricola, but, as already noted, the sand used was always carefully examined, in a manner that entirely excluded the presence of such intruders, before being used for the cultivations. Moreover I have, on more than one occasion, made successful cultivation in which the sand as well as the water used for dilution of the infected fæcal matter was sterilised by boiling.

That experiments conducted in this way do not succeed so uniformly as those in which the sand has merely been previously examined by the biological test of moistening and examining on several successive days, and the water simply filtered, is probably owing to the destruction of bacteria suitable to bring about the decomposition of the fæcal matter surrounding the eggs, as the rhabdites of the ankylostoma can thrive only in decomposed fæces. As to the second objection, I can only say that the absence of Anguillula stercoralis, that is, of the embryos of Rhabdomena intestinale, was carefully noted in the case of each specimen of dejecta used for experiment. Had the so-called Anguillula stercoralis been present it is inconceivable that it should have been overlooked in any examination that detected the presence of the ova of ankylostoma.

Assam is a paradise for the helminthologist, and I was therefore greatly surprised at never once meeting with this particular human parasite in any of the many hundreds of examinations that I made of human dejecta. Further, at the same time as I was conducting these observations of Ankylostoma rhabdites I did a good deal of general helminthological work on animals, detecting many parasites at least as insignificant as the Rhabdomena intestinale, and had that parasite been present in the duodenum it could not have been overlooked in the specially minute examinations I made of the human intestine in all cases in which a necropsy was obtainable.

It is true that the adult parasite (Rhabdomena intestinale) was not known at the time of my investigation, that the embryos (anguillula) had been met with in Africa and in China, and being anxious to compare them with my Ankylostoma rhabdites, I was keenly on the look-out for them; but they did not exist in the parts of Assam I visited, nor have I heard that they have been met with by any other observer in any part of continental India, though I gather that Macdonald has met with them in Ceylon.

In addition to this it is to be noted that it is stated that Anguillula stercoralis can only mature in foul water, and rapidly dies in decomposing fæces. The rhabdites of ankylostoma, on the other hand, mature rapidly and best in the decomposing fæcal matter which is their natural habitat. Up to the time of my leaving Assam I had never once succeeded in bringing them to maturity in water; but since then, when repeating my experiments at Saharanpore in the N.W.P., I succeeded on one occasion in rearing them in water containing a considerable amount of fæcal matter. From these considerations it is evident that these suggested sources of fallacy are entirely excluded by the method of observation adopted, and I may add that the mere fact of their being suggested shows that my critics had not been at the pains to read my report with any approach to attention. As a matter of fact, all these three nematode forms under discussion do very closely resemble each other, though doubtless if one could place them side by side it would be easy to find specific differences; for example, the males of the Ankylostoma rhabdites and of Rhabdites terricola have a caudal membrane supported by seven or eight ribs, but Sonsino's figures of mature anguillula do not show any such structure. This, however, may be merely a matter of careless drawing, or, as I should judge from the figure, the selection of a not quite mature specimen, as this structure only appears in the least ecdyses. As a matter of fact, however, any other life history for ankylostoma is becoming increasingly improbable.

Ralliet has demonstrated a similar dimorphism in Strongylus strigosus and in S. retortie-formis, while I myself have done so in two other sclerostomes, and am strongly inclined to suspect that a large proportion of the so called free nematodes will ultimately be demonstrated to be nothing more than dimorphic representatives of known parasites.

On no occasion have I found these rhabdites in drinking, or, indeed, in any natural collection of water. On the other hand, the fouled soil round infected villages and among the crops, where the natives resort for purposes of Nature, teems with them, and hence I am inclined to believe that soil carried to food by unwashed hands is the commonest vehicle of infection. In this connection I am inclined to think that it matters not whether the embryo

swallowed is the progeny of a parasite or free form, provided only it be swallowed when young. The progeny of both, in other words, develop into parasites or rhabdites, according to the environment in which they find themselves.

Prolonged exposure to the direct rays of the sun kills the free-stage worms, probably because they are thus often brought to a temperature exceeding 140° F., for the prolonged desiccation of even a Punjab summer will not do so, as after a lapse of over twelve months some of the cultivations I had brought from Assam with me to the Punjab were easily revived by moistening; and on being fed with normal fæcal matter were through several generations easily brought to maturity. Burying the embryos also destroys them, and they are easily killed by most of the ordinary disinfectants, such as perchloride or carbolic acid in moderately strong solution.

I will not take up your time by discussing the clinical side of the question, but will merely express a hope that some of those present at this meeting from various parts of the world will give us the benefit of their experience as to the harmfulness of the parasite. My own impression is that it is responsible for a formidable mortality and even greater amount of chronic sickness wherever it exists, but a small group of medical men in Assam are inclined to regard this view as, to say the least of it, exaggerated, while their leader, Major Dobson, of Dhulia, regards the worm as rather beneficial than otherwise—in fact, a sign of robust health.

Dr. Dobson's duties for many years included the examination of large numbers of coolies coming from various parts of India. I had myself demonstrated to him the then new fact that a very considerable proportion harboured the ankylostoma. I also did my best to show him how to diagnose such cases by means of the microscope, but mistrusting an instrument with which he was unfamiliar, he adopted the somewhat heroic course of dosing all comers, healthy and sickly, with thymol, and then examining their dejecta for worms, and subsequently to my visit demonstrated what might have been easily predicted by anyone familiar with the general phenomena of helminthology, namely, that the worm was present not only in those who exhibited perceptible symptoms, but also in a large proportion of the really or apparently healthy.

My object in instituting these examinations of immigrants at Dhulia was to ascertain if the disease existed in the coolies brought from other parts of India, because if this were the case it would explain the fact that the appearance of kala-azar and beri-beri in Assam is coincident with introduction on large scale of the system of imported labour. The fact that the disease existed amongst these immigrants sufficed for my purposes. I therefore examined only such cases as presented some symptoms of ankylostomiasis, though necessarily none were at all advanced cases, as the labour recruiters knew too well that no obviously sickly coolie would pass Major Dobson's critical eye.

Wherever this disease is common, it necessarily follows that a large proportion of the healthy population will harbour the worm also. The presently healthy people may be either severely-infected early cases or slightly-infected cases of any duration; but overlooking the fact that the men he saw had already been selected because they were healthy, and that on this account no really advanced cases could possibly come before him, Major Dobson fell into the fallacy of triumphantly proclaiming to the world that the presence of ankylostoma was a sign of health rather than disease.

Added to this, I am convinced that the wish to escape the disagreeable and almost insoluble problem of the introduction into Assam of measures of "conservancy" which obviously alone can be of any avail in preventing the disease has much to say in the genesis of the thought that these worms are after all harmless.

A second point on which our knowledge of the subject is incomplete is the way in which the parasite brings about the characteristic anæmia. It may, I think, be conceded that the mere loss of blood is insufficient in the majority of cases, only when enormous numbers are present can the drain be adequate.

Personally I am convinced that dyspepsia due to the damage brought to the mucous membrane (a point which I verified *post mortem*) has much to say in the matter, but a further hypothesis is that the parasite produces a poisonous excretion. So far as I am aware, no one has as yet advanced proof of this; but it has been established for certain other entozoa. Lastly, the experience of any gentlemen who may have met with the disease in non-malarious localities would be specially valuable and interesting.

II.—Captain C. F. Fearnside, I.M.S., Superintendent of the Central Prison, Rajahmundry.

A systematic search for the ova of this parasite amongst the convicts and new arrivals in the Central Prison, Rajahmundry, was commenced in February last. Those examined came from the Northern Circars (Ganjam, Godaveri, Vizagapatam, Krishna), also Kurnool and the hilly tracts of the East Coast. The microscopic examination of the stools of new admissions was carried out within a few days after their arrival, and on an average three slides were carefully gone over.

At the end of a year it is intended to publish the full results, and the information furnished is still incomplete. From February 22 to June 18, 1900, 678 new arrivals were examined. In addition to these, more than 300 convicts, who have been confined over six months in the prison, have also been scrutinised. Having a limited time at my disposal it was only possible, as I mentioned before, to examine three slides. So I feel certain that had more time been devoted to the work the percentages would be higher.

Of the 678 new arrivals, 462, or 68·1 per cent., harboured this parasite. Of 100 of these, taken haphazard between April 20 and May 3, the percentage affected is 72, a figure somewhat higher than that of the total 678 persons. The percentage of round worms is the same—36—and thus, for the purpose of comparison, the 100 cases may be taken as a standard. Of the 72 persons affected with ankylostoma, 50 (or nearly 70 per cent.) were in good health, 12 (or 16·6 per cent.) were in indifferent health, and 10 (or 13·9 per cent.) were in bad health. Thus 72 per cent. of persons in the Northern Circars harbour this parasite and remain in good health. Nearly 35 per cent. harbour in the bowel simultaneously both Ankylostoma duodenale and Ascaris lumbricoides.

It is interesting now to compare what are the effects on these entozoa of better hygiene and cleaner food in our prisons. I have attached the analysis of 200 convicts who have served over six months in prison. The percentage of ankylostoma has dropped from 72 per cent. to 58 per cent., and that of Ascaris lumbricoides from 36 per cent. to 18·5 per cent. When the inquiry is finished at the end of a year, I am of opinion that the results will be much the same as those just mentioned.

Of the 105 *post-mortem* examinations made by me in the prison, 74·3 per cent. revealed the presence of this worm; 57·9 per cent. showed congested areas, from one to several centimetres in diameter, in the bowel; and 11·4 per cent. disclosed small erosions and ulcers about 1 mm. to 2 mm. in diameter.

These figures, as well as the experience gained in the gaol after three and a-half years, go to show that the effects of the ankylostoma are for the most part secondary and not primary. They seldom occur in such numbers as to cause true ankylostomiasis, are not the result of the ankylostomiasis, are not the result of the ankylostoma primarily, but of such disorders as malaria, dysentery, &c. The presence of ankylostoma in malarial or other cachectic states is of great importance. It bleeds the patient who can ill afford to lose more blood; it sets up local congestions and erosions of the bowel which cause a catarrh and thus retard the proper assimilation of the food and recovery of the patient.

It is this secondary effect of ankylostoma that I look upon as most injurious.

A word now about thymol. Many are enthusiastic about this drug, and think that its acts like a charm. Observers speak of washing the stools and finding hundreds of dead ankylostomes. I have seen dead ankylostoma in the bowel, and it is by no means easy to say that they are dead ankylostoma, so like are they to decomposed tissues of meat, vegetables, or husk of grain. I do not trust this form of investigation of stools by washing them, &c. I prefer the simple plan of microscopic examination of the motions a few days after the administration of the drug. I attach thirteen cases who have been consuming large doses of thymol for varying periods from ten to sixty days, who were frequently purged, and who at the end had as many ova of ankylostoma in their stools as at the beginning.

For the diagnosis of ankylostomiasis, therefore, it is necessary to exclude all other blood-destroying diseases rather than depend on the mere presence of the parasite in the bowel.

TABLE I.—*Analysis of* 678 *Convicts whose Motions were Examined on Arrival from February* 22 *to June* 13, 1900.

	Ankylostoma Duodenale	Ascaris Lumbricoides	Tricocephalus Dispar
Number of cases in which ova was present	462	245	51
Percentage	68·1	36·1	7·3

TABLE II.—*Analysis of* 100 *Convicts whose Motions were Examined on Arrival from April* 20 *to May* 3, 1900.

	Ankylostoma Duodenale	Ascaris Lumbricoides	Trichocephalus Dispar	Ankylostoma Duodenale and Ascaris Lumbricoides associated in the same Individual
Number of cases in which ova was present	72	36	12	25

TABLE III.—*State of Health of* 100 *Convicts in whose Motions Ova of Ankylostoma were found.*

	Good Health	Indifferent Health	Bad Health
Number of cases in which ova was present	50	12	10

TABLE IV.—*State of Health of Convicts unaffected by Ankylostoma Duodenale.*

	Good Health	Indifferent Health	Bad Health	Number unaffected by any Parasite
Number ..	14	10	4	17

TABLE V.—*Analysis of* 200 *Convicts who have served Six Months and upwards.*

	Ankylostoma Duodenale	Ascaris Lumbricoides	Trichocephalus Dispar
Number of cases in which ova was present	116	37	10
Percentage	58·0	18·5	5

TABLE VI.—*Post-mortems carried out in Rajahmundry Central Gaol.*

Cause of Death	Number of Deaths	Ankylostoma duodenale	Hæmorrhagic Spots	Erosions
Dysentery	29	25	18	8
Diarrhœa	12	11	7	8
Ague	14	2	9	2
Pneumonia..	17	14	10	2
Tubercle of lungs	8	7	5	2
Valvular disease of heart	5	4	3	1
Disease of liver	3	2	2	1
Disease of kidney	13	11	9	1
General diseases	4	2	2	1
Total	105	78	65	12
Percentage	105	74·3	51·9	11·4

TABLE VII.—*List of Convicts Affected with Ankylostoma, Treated by Thymol and Occasional Purgatives.*

Number of Convicts	Period Treated by Thymol	Dose of Thymol Daily	Remarks
994	10 days	20 grns.	Ova as numerous at the end of treatment as at the beginning.
970	17 ,,	20 ,,	
9,635	27 ,,	20 ,,	Ova still numerous.
767	24 ,,	20 ,,	
1,305	15 ,,	30 ,,	
9,708	30 ,,	20 ,,	
629	30 ,,	20 ,,	
774	30 ,,	20 ,,	
743	30 ,,	20 ,,	
9,708	30 ,,	30 ,,	Second period of treatment — Ova still numerous.
9,711	30 ,,	30 ,,	
774	30 ,,	30 ,,	Second period of treatment — Ova still numerous.
743	30 ,,	30 ,,	,, ,, ,, ,, — Ova still numerous.

(To be continued.)

News and Notes.

A PLAGUE SCARE IN BEYROUT.—The special correspondent of the *Medical Record* (September 1, 1900), Beyrout, gives an amusing account of a scare caused in Beyrout by a report that a sudden outbreak of plague had occurred. It would appear that some four persons were taken suddenly ill after a hearty meal and were stricken down with vomiting, diarrhœa and collapse. A rabbit was inoculated with the blood of one of the patients, and after a few days the rabbit died. Immediately a stampede took place, some 25,000 persons left the city in one day, and all communication with the city by sea and land was stopped. There is no proof, however, that the cases of illness were due to plague, and no further spread has taken place. The episode is instructive, as showing what a Turkish community would do in the event of an outbreak of plague; it affords proof also of how plague is spread by refugees, and also how difficult the diagnosis of plague is when signs and symptoms are the only evidence.

LEPROSY IN CRETE.—A medical inspection of the island of Crete reveals the fact that there are 380

known lepers in the island, but the actual number is believed to be double that figure. A leper asylum is contemplated for their reception. It is interesting to note that although lepers have been allowed to roam about at their will, their numbers are stated to be rapidly decreasing.

DESTRUCTION OF RATS BY THE ADMINISTRATION OF BACTERIAL CULTURES.—From the bodies of field-mice suffering from a spontaneous epidemic, Danysz has prepared a culture of virulence sufficient to destroy rats. By a process of subcultures in broth alternating with injections into mice, a highly pathogenic culture was obtained, which, when smeared on bread and laid down for rats to eat, speedily cleared the sewers in Paris of the vermin.

THE ETIOLOGY OF TROPICAL DYSENTERY.—Flexner, in the *Philadelphia Medical Journal* for September, 1900, concludes his remarks on the causes of tropical dysentery as follows: (1) No bacterium has an especial claim to be considered as the predominant micro-organism in the cause of tropical dysentery; (2) no constant bacterial occupant of the intestine, except where dysentery prevails in an endemic form, can be regarded as the probable cause of the disease; (3) it is improbable sporadic and epidemic dysentery are produced by the same organic cause; (4) although the Amœba coli is found in the intestine in health, and in diseases other than dysentery, its pathogenic action in tropical and sporadic dysentery is not thereby disproved. It is possible that bacteriological associations have direct influence upon the pathological powers of the amœba.

THE SURGERY OF HYDATID CYSTS OF THE LIVER.—Jonnesco, of Bucharest, summarises the operations practised for hydatid cyst as follows: (1) Puncture, which may or may not be followed by parasiticide injections; a means of treatment which is mentioned merely to be condemned. (2) Marsupialisation; this is an operation of necessity in the case of suppurating cysts, and in those in which calcification of the wall does not allow of retraction. (3) Incision followed by evacuation and suture of the cyst; the cyst, after being emptied of its contents and germinal membrane, is sutured, and the pouch left is neither drained nor fixed to the abdominal wall. This is the usual procedure followed by Jonnesco. (4) Enucleation and extirpation of the cyst, although the ideal plan, is seldom applicable.

Letters, Communications, &c., have been received from:—

B.—Dr. A. Boddaert (Ghent); Dr. Kailas Chander Bose (Calcutta); Surg.-Major W. J. Buchanan (Bhagalpur).
C.—Dr. James Cran (Belize); Col. Cayley (Weybridge).
D.—Dr. A. B. Dalgetty (S. Sylhet).
E.—Dr. F. G. H. Edwards (Durban); Dr. A. T. Elliot (Assam).
H.—Dr. F. Harrison (London).
L.—Dr. David Landsborough (Kilmarnock).
M.—Dr. Macnicol (Kalua).
S.—Dr. Bassett Smith (Gosport); Dr. Hugh S. Smith (Assam).
Z.—Dr. Karl Ziemann (Wilhelmshaven).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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1.—All communications will be acknowledged in the JOURNAL under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

CONTENTS.—NOVEMBER, 1900.

Original Communications.

SOME DIFFICULTIES IN DEALING WITH LIVER ABSCESS AND HOW TO OBVIATE THEM.

By Neil Macleod, M.D.

Shanghai, China.

An account of the difficulties met with in over a twenty years' experience of the treatment of liver abscess, and the methods of dealing with them, may be of interest to the readers of this Journal.

In 1876-77, during a trip round the world, I made acquaintance with these abscesses in Australia and India, when the routine treatment was by aspiration. In January, 1878, in conjunction with Dr. Henderson, of Shanghai, I had the opportunity of treating by incision and free drainage with the Listerian precautions of that time, what we believed to be the first recorded case[1] of liver abscess dealt with in that way. Nothing could have been more satisfactory. In 1879, I had to deal with three cases, in one of which somewhat startling hæmorrhage from the incised liver tissue, to reach a deep-seated abscess, led to my discarding the knife and to the use of a fistula forceps passed down by the side of the aspirator canula, the blades being then opened and withdrawn. In 1884, in the case of a lady, I opened and drained an abscess of the dome and back part of the right lobe from the side through an intercostal space, without (or with very feeble) adhesions. The drainage tube slipped out the night after operation, during an attack of vomiting. On attempting to replace it, solid liver substance was found opposite the opening in the wall. After some trouble the cavity was found and the tube reintroduced. The track became very oblique, causing much annoyance in replacing the tube as cicatrisation proceeded. This case recovered, but in 1885 a similar one, also a lady, seen in consultation, the tube came out several times in the intervals between changes of dressings, in spite of various precautions taken to prevent its escape. Each time an anæsthetic had to be given before the cavity could be found and the tube replaced. This case died, it must be confessed, in consequence of failure of drainage. The *post-mortem* examination revealed a single abscess cavity. The tube track was through the pleura, and at its point of passage through the diaphragm, there was some thickening, and the tube had a kink. When the contents of the cavity had been above a certain quantity, they flowed readily, but below this quantity with difficulty.

This experience led me to substitute for the rubber drainage tube a metal one which could not kink and could be held more readily in position. An oval tube would obviously be of advantage for use between the ribs. Before my metal tubes were ready another case presented itself. The day after the operation, the discharge being too scanty, and the tube appearing not to be in the cavity, an anæsthetic was given, the cavity searched for and found close to, but shut off from, the track already made, half a pint of pus escaping. Either the drainage tube had not entered the abscess the previous day, or it sank into the soft wall which often surrounds these abscesses. It is evident that if the aspirator trochar lies in contact with the boundary of a cavity, the usual larger operating one introduced in a slightly different direction, though at the same opening in the skin, may easily fail to enter the abscess. Later, in the conduct of such a case which has been entered close to the wall of the cavity,

[1] *Lancet*, vol. i., 1878.

as it contracts, the track of the drainage tube will become more and more oblique. I have seen the direction of the drainage tube change considerably, even during evacuation, at the operation. These experiences and considerations suggested the desirability of determining the relation of the point of entrance of the aspirator needle to the boundaries of the cavity and its attainment, by sounding through the aspirator canula before evacuating. This object I attained by passing a knitting needle through the canula into the abscess, to ascertain depth, position of boundaries in various directions, which furnish data for an approximate opinion as to the size and position of the abscess, its relation to the point of aspiration and the probable future behaviour of the drainage track made at that point. Leaving the knitting needle in position, a more desirable point of aspiration could be tested if that seemed necessary. If the aspirator canula is withdrawn, leaving the knitting needle in position, it serves as a guide for the larger operating trochar and canula one-eighth of an inch in diameter. Withdrawal of this trochar being followed by pus, and the knitting needle removed, a grooved guide, fitting the canula and 10 inches long, is next passed into the cavity and the canula slipped out over it. The groove guides the dilator point into the abscess.

The metal tubes were first made for me in 1896, by a Chinese silversmith (and later, by Messrs. Young, Forrest Road, Edinburgh), in four lengths, 4, 3½, 2½, and 1½ inch of oval calibre $\frac{3}{10}$ by $\frac{4}{10}$ inch (I now use ½ by ⅜ inch), both ends open, one of them having in addition two large openings in the side to facilitate drainage, the outer end being pierced by four small holes, through any opposite two of which a safety pin is passed and prevents the tube slipping below the skin level. Examination of the normal intercostal spaces in twelve bodies showed that tubes of these dimensions can be easily passed at any point where it is desirable to operate. To render their introduction easy, both at operation and at change of dressings, I had made at the same time a pilot (see fig. 3b) 4¾ inches long, flanged at one end, the extra ¾ inch forming a conical point which projected that distance beyond the end of the longest drainage tube of the set. The ends of the pilot are open for the passage of the grooved guide, over which the drainage tube, with the pilot inside, easily slips into the abscess. The withdrawal of the guide and pilot leaves the drainage tube in position. Subsequently, when it is necessary to withdraw and cleanse or change the tube, the grooved guide serves for determining how far the cavity has contracted, when the tube can be replaced as before. This may seem unnecessary trouble in the introduction of a drainage tube, but those think otherwise who have had to deal with oblique sinuses draining abscesses in a liver which has changed its position since operating and is recovering its movements with respiration.

Subsequent consideration made it evident that no more damage to the tissues torn than is necessary for the introduction of the drainage tube would be an improvement. It was difficult to limit the damage done to that extent with a forceps. In 1890, I had made a straight-bladed dilator on the principle of that of Lyon's for the urethra, the parallel blades of which are separated by means of a screw, so as to distend a loop of tissue equal to the circumference of the drainage tubes. The points and blades of the dilator are of the same thickness as those of the fistula forceps, the whole being 10 inches long. In the case of the first dilator supplied by the maker, it failed to open to the desired extent, but one turn on its axis sufficed to attain what was wanted—the easy entrance of the drainage tube. The latest form (see figs. 1 and 2) of dilator has a stop to allow of variation in the degree of dilatation and the use of the dilator for other purposes.

Unless the abscess is pointing, and even then, it is well to make the absolute diagnosis with the aspirator under an anæsthetic, practically commencing the operation therewith. The following precautions with the aspirator will save both time and trouble: the aspirator, canula and tube should contain no fluid. Carbolic solution and blood make a mixture not unlike liver pus. When repeated aspirations are made, the tube and canula should be cleansed and emptied before each aspiration. The quantity aspirated should only be sufficient for diagnosis, and it may be necessary to inspect the material after ejecting it from the canula and tube into a vessel. There should be a glass tube window inserted in the aspirating tube *close* to the canula. The canula with spear pointed trochar is better than a hollow needle, which, if large enough, is apt to punch out a piece of tissue, and if small, may be easily blocked by the necrosed masses so frequently occurring in these abscesses. If no adhesions are present, the punching out of a piece of liver tissue, as described, increases the risk of escape of abscess contents by the track of the needle when it is withdrawn, the escaped matter being smeared over the peritoneal wall opposite, as the liver moves up and down with respiration, an objection raised by some to the use of the aspirator. Both escape of contents and movement of the liver are prevented in such a case by the use of a spear-pointed trochar and the withdrawal of the canula, leaving the knitting needle in its track.

Since 1888 I have used for suction what is known in America as Allen's surgical pump. It is certainly the simplest, most ingenious, workmanlike and compact means of aspirating ever devised. It can be used either as aspirator or, by reversing the handle, as an injector. There is no bottle, piston or valve to go wrong. The only part of it that can fail is the rubber tube, but I may say that the one supplied with the machine acts efficiently now after eleven years' use in the East! the spare one never having been needed. The pump only should be purchased. I cannot recommend the needles supplied. Any set of trochars can be used.

By these means I have endeavoured to secure the objects which one should have in view in dealing with these abscesses, viz.: —

(1) Aseptic, free drainage of their contents.

(2) To provide the shortest drainage track emerging at a point in the abdominal or chest wall, preferably in front or laterally.

(3) If the liver is not adherent to the wall of the abdomen at the point of puncture, to prevent leakage of the contents into the pleural or peritoneal cavities,

and to maintain opposite each other the openings in the organ and wall during the operation.

(4) To anchor the liver securely at that point, and create adhesions if they are absent.

(5) To do no more damage to the soft and bony tissues than is absolutely necessary to secure these ends, with as little hæmorrhage as possible.

(6) To keep the drainage track open until the abscess cavity has contracted all round the drainage tube and discharge has ceased.

Manson's very ingenious method of operating secures several of these points, but, inasmuch as his tube must be very thick walled to stand the strain of stretching, as it is closed at the end in the abscess by a metal plug, drainage being by one or two holes in the wall, cannot be so free as through an open ended, thin walled, metal tube, also with side holes. Being of rubber, his tube may kink and give rise to the difficulties above described. In addition, Manson makes no provision for his second trochar entering the abscess by the track of the first when no adhesions are present, affording a possible danger of leakage quoted by the opponents of aspiration, even for diagnostic purposes. The manipulation of Manson's apparatus requires some dexterity and practice as witnessed by an occurrence in the hands of a surgeon I saw using it, and that not for the first time. The drainage tube having been introduced into the abscess through the canula, the steel stilette, on being released, was shot out of the liver forcibly against the ceiling of the room by the tensely stretched rubber tube, past the side of the surgeon's head as he bent over the table. One could not help thinking how awkward it would have been if the inner end of the drainage tube had given way, the stilette driving the metal plug into the patient instead. I have several times seen the rubber tube give way at the openings in its side when being stretched and fixed to the stilette.

The *certainty* of the presence of adhesions between liver surface and abdominal wall, can only be attained by opening the peritoneal cavity. Frequently they can be located with a sufficient degree of probability at the point of acute stabbing pain referred to in the previous history, rarely by hearing or feeling friction, very occasionally by inflammatory signs of the abscess involving the skin or subcutaneous tissues. With adhesions presumably present at a suspected point, to open into the peritoneal cavity would obviously be a mistake. Even if adhesions are believed to be absent, that is, if the surface of the liver is not involved and this is found to be the case on exposing the liver, what can the operator do on finding nothing visibly or palpably indicating an abscess? He must either aspirate or give up the quest, unless he is prepared to continue the search by further incisions.

It is claimed by some that adhesions can be best secured by stitching the liver to the abdominal wall previously incised. Manson, in his work "Tropical Diseases,"[2] represents me as considering "that stitches will not hold in the soft and inflamed liver tissue." This scarcely represents my opinion on this point. When the inflammatory process has not involved the liver *surface* and adhesions are therefore absent, the liver capsule is then normal and too thin to hold stitches in the moving organ; whilst if thickened by the inflammation, the capsule will be *adherent*, not needing to be stitched. I tried to stitch a normal liver to the wall in a dead body, but found that the stitches when tightened cut through the capsule and liver tissue so easily that I did not feel encouraged to attempt to stitch the organ disturbed by respiration and possible vomiting, or other violent movements. It has to be remembered that these stitches are proposed to be passed before the abscess is opened. An incision opening into the peritoneal cavity encourages the entrance of air between the liver surface and the wall, and therefore may promote the access of discharge to the same region.

I have seen only one case where an abdominal exploration would have been of service, but in it the exploration would have been *after* the evacuation and drainage in the ordinary way, because of improvement not following free drainage. The history of the case is of interest: a visitor to Chefoo had acute liver disturbance after an attack of acute dysentery. The aspirator was used, but no pus found. The patient shortly after sailed for Shanghai, and on the voyage symptoms of collapse manifested themselves, indicating rupture of an abscess or of an intestinal ulcer into the peritoneum. I was inclined to the latter belief, as acute pain had been felt suddenly in the region of the descending colon and continued till I saw him two days later. Within a few days I emptied an abscess in the left epigastrium at a point close to the middle line. The abscess drained freely, but the patient died of exhaustion. On *post-mortem* examination I expected to find multiple abscesses. There was, however, below the left lobe and shut off from the rest of the peritoneal cavity, only a second abscess communicating by a small hole with the one opened and through which the second was draining.

When operating through the chest wall some surgeons recommend the removal of a piece of rib; first, so that the layers of the pleura can be stitched together, and that the liver can be fixed to the diaphragm. The objections to the latter proceeding have been already stated. If the pleural surfaces are not adherent this means that a moving diaphragm has to be stitched to the chest wall, and that the lung will collapse, encouraging leakage of pus into the pleural cavity. I have seen this happen, but I have never seen a lung collapse on the pleura being pierced with my tubes as described. Second, for the purpose of securing better drainage than when a piece of rib is not removed. Anyone who is of this opinion need only inspect the intercostal spaces from the inside of the chest, to see that without interfering with ribs, there is abundant space between them for the passage of sufficiently large tubes at any desirable point. In the case of hydatid cysts of the liver it may be necessary to remove a portion of rib to make an opening large enough to allow the escape of large daughter cysts, but in liver abscess where there is no reason to provide for the escape of such bodies, the damage to ribs and the surrounding soft parts seems to me absolutely unnecessary, and an unjustifiable addition

[2] "Tropical Diseases," p. 372.

to the patient's risk, criticism which may be applied with equal force to laparotomy in these cases.

Operation.—Every means having been taken to secure asepticism, and pus having been found by aspiration, a knitting needle or similar long probe is passed through the aspirator canula into the abscess; its size and position in relation to the entrance thus made, are determined by tilting the probe in various directions and to different depths. Having thus settled that this or some other is the best point for drainage, the aspirator canula is withdrawn over the probe left in position. The operating (larger) trochar and canula are next passed by the side of the probe, the trochar withdrawn and if pus flows from the canula the probe also is withdrawn. Through the operating canula the grooved guide is passed down into the abscess and left there, the canula being withdrawn over it. A straight incision of an inch in length is made through skin and subcutaneous tissues,

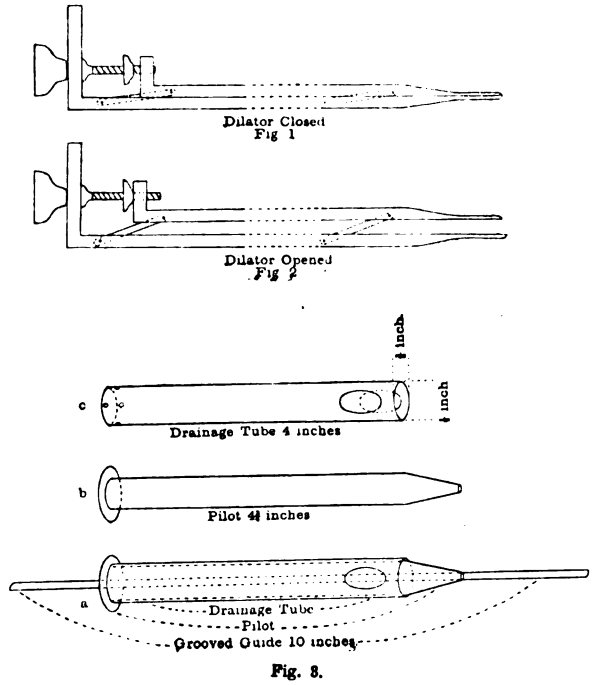

Fig. 3.

the guide being in the middle of it. The closed dilator (fig. 1) is next passed along the groove into the cavity, the blades opened to the requisite extent and the dilator then withdrawn, the guide still remaining behind. One of the four drainage tubes of suitable length with the pilot in its interior is threaded on to the guide (fig. 3, a) and so directed into the abscess. The guide and pilot are now withdrawn, leaving the drainage tube in position, and a safety pin fixed in the projecting end. When the abscess contents appear to have ceased flowing, with the aid of gentle pressure on the abdomen, the dressings can be applied.

The above procedure takes less time for its execution than for its description, and affords an efficient and easy method of operation.

NOTES FROM LAGOS, WEST AFRICA.

By Henry Strachan, *Chief Medical Officer, Lagos.*

NOTE XIII.—Anopheles and Malaria at Badagry.

In Note XI. I mentioned the interesting fact that a place called Badagry, in Lagos, had been notably free from malaria cases (new infections) for some months, though its environment was eminently favourable to the existence of the malaria parasite, and that during those months (amounting to nearly three years) I had been unable to procure or find any Anopheles. Yartha pointed out that at any time malaria might re-appear in that town (for persons having latent parasites in their blood are constantly going thither) and that it would be interesting to note whether a burst of malaria would be accompanied by the presence of Anopheles, which abound a very few miles away. A few weeks ago such a burst of malaria did occur. Five or six persons were attacked almost simultaneously, three of them being daily companions of one another. I, as soon as possible, procured specimens of mosquitoes from the place, and found amongst them, for the first time, several Anopheles.

I therefore record this as a supplement to Note XI.

November 3, 1900.

THE TREATMENT OF NIGHT BLINDNESS BY INGESTION OF LIVER.

By Major W. J. Buchanan, I.M.S., M.B.
Bhagalpur, Bengal.

The affection known as night blindness is, I suspect, a sufficiently common complaint in all tropical regions to make a note on a very simple and effective method of treatment of interest to the readers of this Journal.

The method of treatment here to be described was first brought to my notice by Major F. P. Maynard, I.M.S., in the *Indian Medical Gazette* for June, 1900. The treatment by ingestion of liver is as old as Hippocrates; it is well known to the native practitioners of some parts of India, and it appears from the article by Professor Trantas, quoted in a recent number of the *Ophthalmic Review*, to be equally well known as a domestic remedy in Greece.

I have within the past few months treated some twenty cases of genuine night blindness with great *and immediate* success and consequently think it right to call the attention of practitioners in other parts of the tropics to it. I would also ask any medical men who try it to let me know through this Journal if the method is known in the part of the world where they practice.

The treatment is simplicity itself: it consists in giving about 8 ozs. of liver (goat's, sheep, or ox), fried in oil, and with spices, daily for a few days. Generally it will be found that five or six days is sufficient to effect a cure, but I have usually continued it for at least a week. Even as soon as the second day some improvement will be noted, and the cure is usually complete by the seventh day. I have never given any other form of treatment with the liver. We all know that cases of night blindness have a tendency to get

well of themselves in the course of time, but under what is called tonic treatment and ordinary dieting, such cases will not be even improved under two or three months. When I first commenced this treatment the results were so immediate and so satisfactory that I became sceptical of the reality of the previous complaint, and I wrote to several friends in India to try the liver treatment, and in every case in the course of a few weeks I got letters to say that the liver treatment had met with equally satisfactory results at their hands. I have not yet had a single failure with it, nor any relapse on cessation of the treatment. As liver-and-spices is by no means an unpalatable dish, and consequently appreciated by the patients, it will be found necessary to be very sure that one's cases are genuinely night blind. In all my cases I took special trouble to make sure that they were genuine before putting them on the treatment.[1]

At present I am endeavouring, at the suggestion of a friend, to make a glycerine extract of liver, the use of which would not lead to pretences of night blindness as the palatable fried liver does. I have been told that cod liver oil with a few minims of turpentine acts in somewhat the same way.

It seems to me probable that the liver in these cases acts somewhat after the fashion of thyroid and suprarenal extract, and the other substances used in organotherapy. The immediate good effect cannot solely be due to any nitrogeneous element in the food, for an equal quantity of fresh meat is useless.

In conclusion, I can confidently recommend this simple and effective method of treating a very unpleasant condition.

CEREBRO-SPINAL MENINGITIS.

By E. G. Hamilton Williams, D.P.H., M.R.C.S., L.R.C.P., and Mary Hamilton Williams, M.B., B.S.Lond., D.P.H.Cantab.

Special Service Medical Officers, Ashanti Field Force.

The following is an account of an epidemic of cerebro-spinal meningitis which occurred among a number of carriers brought from Mombassa, East Africa, to Cape Coast, West Africa, for duty with the Ashanti Field Force :—

These carriers came in three transports. The first transport brought some 1,500 carriers, who are described as having been of poor physique, and of whom about 200 were landed sick. They suffered much from pneumonia and dysentery, and had many deaths on the voyage, but there appears not to have been any cerebro-spinal meningitis among them.

The second transport brought about 1,500 carriers, who, as a body, appeared to be a serviceable set of men. About 30 were landed sick, suffering from various complaints, and were sent straight to the Base Hospital. Of these we noticed two the same evening to have retraction and rigidity of the head. They would seem to have been the first cases of cerebro-spinal meningitis noticed at Cape Coast.

When the blood of these cases was examined some days later by us, it was found to contain diplococci, both intra- and extra-cellular, and micrococci. Fresh cases cropped up from day to day, till altogether some 30 cases were identified. These fresh cases, with three exceptions, all occurred among East African carriers suffering from other diseases, who were quartered in the Roman Catholic School Room, which was used as an overflow hospital for slighter cases from the Base Hospital, which was much crowded.

The Roman Catholic School Room is a brick building, white-washed, with concrete floor, very well ventilated, cool and airy. It is, however, situated in a hollow, and a good deal shut in by surrounding buildings. Most of the carriers spent the day in the compound outside the building. As the cases were diagnosed they were at once removed to the Base Hospital, where half a hut was set aside exclusively for these cases.

This hut is a temporary wooden building with a hard mud ("swish") floor. It was badly ventilated, and is cut off from the prevalent winds by another hut. [Since writing the above eight more windows have been made in this hut, and it is now very well ventilated.] In contrast, however, to the Roman Catholic School Room, this hut is situated on a hill, high above the town, a very fine site.

Of the three cases mentioned as having occurred among other than East African carriers, two were Mendi boys (from Sierra Leone), one of whom had only come from up-country two days before. The history of the second case could not be traced. The third case was a Kroo boy, one of the hospital labourers. As far as is known, he had been in contact with the patients only when he assisted to carry them up to the Base Hospital in hammocks.

An attempt to arrive at an early diagnosis was made by examining blood fibres taken from all the carriers at the Roman Catholic School—about 30 in number. In no case did we find diplococci; yet forty-eight hours later one of these men was found to have marked cerebral symptoms, and on re-examination his blood was found to contain diplococci. This man died within sixty hours.

The following is a description of a patient typical of the larger number of the cases. The patient would complain of pain in the head—generally indicating the temples—and in the back of the neck. The head is retracted and rigid, any attempt to remove it from the position in which it is rigidly held being attended by considerable pain. Temperature 100°. The pulse would be 90. Respiration unaffected. Tongue with a thick white coat, yellow at the back, pink at the edges and tip. Bowels constipated. Within two days severe paroxysmal pain in the head would set in and death would ensue shortly after.

General Appearance.

The disease, *per se*, did not appear to cause emaciation. There were no skin rashes or cutaneous hæmorrhages. No herpes. The facial expression was one of anxiety and pain. The head markedly retracted; the patients refused either to bend or rotate the head, and it was difficult or impossible to make them do so. There was also pain in the back of the neck,

[1] Night blindness due to *retinitis pigmentosa*, a rare disease, is not meant, but the common form so often found in malarial or scorbutic cachexias.

greatly increased by movement. In some cases, however, the head symptoms consisted only of giddiness.

Temperature.—Varied between 100° and 104°, commonly about 100°.

Pulse.—There was nothing characteristic about the pulse. It might be 80, full and strong, with a temperature of 102°, or in another case 110, with temperature 100°.

Respiration.—The rate was unaffected.

Alimentary System.—Tongue generally as above, but in some cases clean. Bowels tend to constipation, which yields easily to treatment. Appetite good until near the end. No vomiting, nausea, or abdominal pain. Urine normal. No albumen.

Nervous System.—Headache, with retraction and rigidity of the head were the most constant symptoms. Out of 30 cases these symptoms have been absent in only 3, and these presented giddiness. Knee jerks were absent or diminished. No hyperalgesia or hyperæsthesia. No tenderness of muscles or on pressure over any nerve-trunk could be detected. No convulsions. Strabismus occurred in 4 cases, all of which died. Amblyopia in 1 case; and there was loss of sight in one eye in another. Both of these patients had recovered when the eye symptoms were complained of. No ptosis or abnormalities of pupils. Right hemiplegia in 1 case (died). In the cases which died in the early acute stage of the disease, the majority retained their intelligence to the end, but several cases, after showing marked improvement, gradually developed either coma or insanity, and died. No deafness was detected in any of the cases. Duration varied from sixty hours to three weeks; most commonly about five weeks.

Blood Examination (from finger tips).—Any blood count was impossible, owing to lack of instruments; but there was undoubtedly an early leucocytosis; the increase being mainly in the polynuclear cells. It was, however, very difficult to judge how far this was due to the original disease, not meningitis, from which the men were suffering.

In every case diplococci were found, sometimes intra-cellular. In the latter case they were either in the polynuclear leucocytes, or in a cell which was somewhat larger than these with a single large round or oval nucleus. In some cells there were more than two cocci. The diplococci are somewhat larger than gonococci, but resemble these. They stain deeply with the usual bacterial stains. From the short description given in "Muir and Ritchie's Bacteriology" of the diplococci found in cases of cerebro-spinal meningitis by Weichselbaum, we judged them to be the same organism, but there is no mention of his finding them in the blood.

Treatment.—The only drug which appeared to be of any service was morphia in large doses. The tendency to relapse or become comatose after apparent recovery was best combated by keeping the patients entirely out of doors.

Several *post-mortems* were made: the following is reported as having the most complete history. A carrier, at the Roman Catholic school, suffering from slight dysentery, was found screaming with pain in the head. He was given morphia gr. j. hypodermically and slept at once. His blood had been examined forty-eight hours previously and we had failed to find diplococci. It had shewn a leucocytosis, in common with many of the dysenteric cases. Fresh films were taken and many diplococci found. He died within sixty hours. Necropsy, four hours after death.

Male aged about 40, body emaciated (from dysentery). No *post-mortem* rigidity. Risus sardonicus well marked. Pupils 4 mm., equal.

THORACIC CAVITY.—Right parietal *pleura* adherent; no recent pleurisy. Trachea and lungs normal.

Heart.—Four ventricles contained white clot.

ABDOMINAL CAVITY. — Marginal engorgement of intestinal vessels. No enlargement of *spleen.*

HEAD.—*Skull* of ordinary thickness and density.

Dura mater.—Sinuses full, vessels engorged.

Pia mater.—Intense engorgement of all vessels, including the smallest capillaries. On the superior surface of the brain, particularly along the mid-line and over the frontal convolutions, there was marked dense, white, thickening along the sulci.

The *sub-arachnoid space* was distended with fluid, in which were flakes of pus.

Ventricles.—No excess of fluid. No granulations.

Substance of the brain.—Shewed on section innumerable vascular points, much larger than normal, from which blood exuded.

On cutting through the medulla to remove the brain, a steady stream of *spinal fluid* poured out of the spinal canal. It was bright straw-coloured and contained fine red flakes, like cayenne pepper (blood).

Portions of brain, spinal cord, and of the various organs were removed for future microscopic examination.

The following were also examined at the time:—

(*a*) Film from the sub-arachnoid fluid: this showed leucocytes in various stages of breaking down. Micrococci resembling ordinary staphylococci and streptococci; diplococci resembling those found in the finger-blood, but mainly intra-celullar; granular *débris.*

(*b*) Film of blood from cranial cavity: red blood-cell; leucocytes, large numbers of polynuclear, and also many large, with large oval or round nucleus; also a few pus cells, and organisms as above.

(*c*) Film from fluid in spinal canal: resembled (*a*), with the addition of a few red blood-cells and hæmoglobin crystals.

(*d*) Film of heart-blood: resembled peripheral blood, but shewed epithelial-like leucocytes.

REMARKS.—In addition to the meningitis cases, similar diplococci were found in the peripheral in those cases of pneumonia in which there were no cerebral symptoms except slight headache. Bearing in mind that a large number of deaths on board the transport were attributed to pneumonia, this is suggestive.

We are unable to offer any conclusions as to the method of spread of the disease. It did not spread where we should most have expected it, viz., in a hut where all the seven cases were placed, and which was so badly ventilated as to remind one of a monkey house, though the other carriers, in defiance of orders, would sleep there. Up to the present time it has not spread to the white people, although some of us have been greatly exposed to infection, if the infection were conveyed in any manner analogous to any of the well-known infectious diseases.

It has appeared entirely unaffected by weather. It began in the rainy season, and continued in the dry weather. The average temperature has been from 70° to 80° F.

We have tried in vain to arrive at data as to the incubation period.

We had believed that cerebro-spinal meningitis was new on this coast, but the Principal Medical Officer of the Ashanti Field Force, writing in answer to our question as to what steps, if any, were to be taken to prevent it spreading, states that he has many officers with him well acquainted with the disease, and that he himself has suffered from it. Whether they have seen it here or elsewhere, we do not at the time of writing know.

* * * *

Since writing the above we have learned from Dr. Hayford that there is a case of cerebro-spinal meningitis in a native living next door to the Roman Catholic school.

Our thanks are due to Dr. S. H. R. v. R. de Groot, S. M. O. Base, for his unfailing courtesy in allowing us every facility to examine and report on cases.

MALARIAL ULCERS IN BRITISH CENTRAL AFRICA.

By David Kerr Cross, M.B., C.M.Glasgow.
Blantyre, British Central Africa.

Malarial ulcers are phagedænic in nature. They are common all over British Central Africa and East Africa generally. It is impossible to give an exact percentage of this appearance amongst Europeans, but probably 18 per cent. of those who have been in Central Africa for a period of two years suffer from these irritating sores.

The typical malarial ulcer is an angry-looking, punched-out, circular sore, covered by a non-transparent pulpy membrane, under which the tissues are ulcerated and infiltrated. The surface of the ulceration is covered with a greyish, smooth, velvety, fœtid material which is a mixture of dead tissue and parasites. Its favourite seat is on the ankles or dorsum of the foot, or indeed any part under the knees; I have seen them, however, on the arms, body, and even the head. Usually the sore is one-and-a-half to two inches in diameter and single. Six or eight distinct sores on the legs below the knees are, however, not uncommon. All spontaneous phagedænic sores have a marked predilection to attack the lower limbs, and out of one hundred cases fully ninety are in these pendant tissues.

Should the sore make its appearance in previously healthy skin, the attention of the patient is called to the spot for two or three days previous to the actual breach of the dermis. It is painful, dry, and swollen, with pale edges. Later on, a little bleb containing watery material forms and bursts, and, if carefully looked for, granulations of a greyish colour are seen, together with the beginning of the formation of a false membrane. This is the usual course of origin, but any breach of the skin such as a mosquito bite on a tissue otherwise weakened may result in a "fever sore." When these blebs are forming and bursting there is usually slight constitutional disturbance; there may even be a slight rise of temperature. With great rapidity the ulcers disintegrate the skin and fascia, and at length become typical sores. In four or five days it may have grown to the size of a five-shilling piece, and if not attended by rest and treatment will soon be inches in diameter. It is now a circular cup-shaped, stinking, greyish slough, with projected hard edges, discharging a watery, yellowish fluid. It continues to grow at the edges while its centre dies, and discharges a fœtid exudation which infiltrates and destroys the surrounding tissue. This goes on for several weeks or months, when the sore seems to lose its active growing properties and to remain stationary. In very severe cases, as seen amongst natives, and especially amongst slaves on the march, not only is the skin and fascia attacked, but the muscles, tendons, vessels and nerves down to the periosteum itself.

Usually one sore only is seen, but six or eight lesser sores may be on the legs as satellites of the one large sore. It is quite possible to see them at the various stages: thus one may be at the stage of irritation and swelling, another may have formed a blister, a third cast off its slough, and a fourth at the resting stage with callous edges. Should the sufferer persist (notwithstanding the pain) in walking about, the skin around the sore becomes markedly congested and swollen and the constitution seriously affected. Yet, on the other hand, I have seen Europeans hobbling about notwithstanding they had offensive sores on the legs as big as the palm of the hand. Such Europeans seemed to suffer in no degree comparable with the size of the ulcer.

In all these malarial sores there is a marked tendency to self-limitation, and also it seems to me that the tissues around are to some extent anæsthetised. The zone round the margin of the sore, bordering on the healthy tissue, and which is of the colour of diluted red wine, is painful at all stages, but when the sore becomes chronic, the raised-up edges are covered with scabs and infiltrated, and consequently lose much of their sensibility.

The fever sores as found in Central Africa may be divided into the following:—

(1) The large sloughing sore referred to above, which begins as a bleb, eats into the tissues, has a pulpy false membrane and a fœtid exudation. It is seen on the lower limbs, may be inches in diameter, and has an indefinite course.

(2) A lesser sore which usually originates in a slight abrasion of skin. It may appear on the upper limbs or hands, or any part of the body indeed. When the skin is broken in those who have a predilection towards such sores, the wound does not heal but developes a slough. It will not heal for weeks or months.

(3) A form which comes in crops, or at least in regular succession. These may appear below the knees and begin as itchy blisters. They seldom grow larger than a shilling and go through the various stages in a fortnight. Sometimes a dozen or more are seen on the legs at one time and in all stages of devolopment. I have seen one of these sores take upon itself a more active growth while the others faded away.

(4) A less formidable form appears all over the body, even on the head. They come as crops of papules going on to pustules about the size of a bean. They discharge a thin watery pus, then dry up and pass away to be followed by another crop.

Malarial sores seldom attack Europeans till they have been a year or two in the country, or until they have had several attacks of malarial fever and have been constitutionally reduced. Yet I have seen sores appear on a European after a few weeks' experience of Central Africa. They do not attack every European. Some constitutions have a predilection towards malarial ulcers, and whenever such persons receive an abrasion, however slight, on the body or limbs, and above all on the hands, that abrasion developes into a respectable ulcer. The slightest cut developes into an ulcer that will give trouble for weeks. An insect bite on the ankle will be the starting-point in some constitutions of a sore that may be inches in size and seriously affect the constitution. On the other hand, it is the universal experience in Central Africa that *a European who suffers from these sores does not suffer from fever, id est,* from malarial fever in the ordinary sense. This is the reverse of what we would expect. All who suffer from these sores assert that when the sores on the legs are open and demanding daily attention they never have fever, but when through rest, dressings and tonics the sore is induced to close, they begin very soon afterwards to have attacks of fever. Whilst this is the ordinary observation that a person who has an open one seldom or never has fever, more careful observation proves to us that, should a sufferer from open sores develop an attack of malarial fever of the remittent type, there is a marked tendency for the sore to grow backwards, as it were, and become formidable in type. It is no uncommon thing, as I can testify, to find, after an attack of fever, that it has increased in virulence and size.

All over Central Africa it is a common thing to find malarial sores on the legs and hands of otherwise strong, healthy men. Such men may even be putting on flesh and may be looking uncommonly robust for Africa. They never have fever, but on making enquiry you find they are unable to walk about with freedom, owing to one or two malarial sores.

I have had occasion to study these sores *carefully* on twenty-nine Europeans in British Central Africa, and on very many natives. On every occasion where the sore was covered by the typical greyish false membrane, which when raised seemed to adhere to the tissues underneath, an examination of the grey pus revealed the same features. If a particle of the greyish membrane be placed between two cover-glasses, dried by means of the spirit-lamp and stained by any of the analine dyes, the same structures will be seen: namely, a swarm of rods, some straight, some bent, but a swarm. They are bacilli, and are doubtless the cause of the sore. Very often two micrococci may be seen. The clear watery discharges which come from the sores also harbour swarms of rod-shaped bacilli.

Believing that the discharge from the typical sore is the cause of its dissemination, I have tried to inoculate several animals, such as dogs, cats, and goats, but have not succeeded. I have even tried it on myself, but with a negative result. I have tried it on natives and only partially succeeded. It seems to me, judging from the fact that only a small proportion of Europeans suffer from these sores, that in addition to the bacilli and micrococci invariably found in the wounds two things are necessary: *first,* a constitutional tendency, and *second,* that constitution must be reduced by some impairing influence, such as malarial fever. Many Europeans during their residence in this country have been reduced in health but have not once suffered from these sores; while others, to outward appearance in health (at least for malarial Africa), had several sores on the lower limbs.

Inoculation is common amongst certain of the natives. I have seen a native who had a typical sore on the leg squatting on the ground and allowing the same to touch a small abrasion on the opposite foot. Indeed, I have encouraged him to do so and watched the inoculation with interest. In seven days the inoculated spot had developed a sore exactly like the original. This same native allowed me to make two little incisions and to inoculate with the discharge. They both became sores but were not equal to the one that had grown naturally.

On the treatment of fever sores a word only is necessary. The first indication is bodily rest in the horizontal position with elevation of the limb. The next is, realising that we have to do with a phagadænic sore and bacilli and dead tissue, we must annihilate these deleterious influences by the actual cautery or other strong caustic, as pure carbolic. I usually give the natives chloroform and scrape away the false membrane and dead tissue and freely swab by carbolic. In a few days a healthy looking sore free from all puridity is the result. The wound is then dressed by one or other of the excellent antiseptic dressings. A creamy-looking powder, made of iodiform one and acid boric three, dusted on these wounds acts like a charm on natives. These measures must always be accompanied with tonics, as iron, arsenic, quinine and strychnia.

CIRCUMSCRIBED CUTANEOUS ŒDEMA.

By H. Campbell Highet, C.M., M.D.
Physician to the Royal Palace, Bangkok, Siam.

The appearance of a second paper with the above heading in the Journal of Tropical Medicine, leads me to publish a few notes on a similar condition seen in Siam, with a hope that further and more definite information may be furnished by some of the many readers of the Journal. I regret, however, that I must write from memory, as I have not my records of cases here with me in London.

The occurrence of localised swellings is by no means an uncommon complaint of one's patients in Siam. Their site varies, as I have seen such swellings on the dorsum of the fingers, hand, wrist, dorsum of the foot, leg, or even on the back. The Siamese simply call the disease "boüm" which being literally translated means *swelling.*

In one or two cases, there has been a slight rise of temperature, but as a rule no fever has been noted by

me in such cases. The swollen part may or may not tingle and itch, it pits on continued pressure, and the skin is unaltered in appearance, except that slight pallor from anæmia of the cutaneous vessels, owing probably to the œdematous pressure, is often seen. The swelling subsides in a few days without treatment. No constitutional symptoms have been observed. In one case, however, while attending a Siamese lady, suffering from simple cystitis, my attention was drawn one morning to two adjacent fingers of one hand, which had been observed to be swollen.

I found this circumscribed œdema present, not only on the two fingers, but also on the dorsum of the corresponding metaphalangeal areas. The œdema gradually faded away over the dorsum of the hand, and did not end abruptly. Recovery took place in a few days without treatment, and nothing more was thought of the œdema. A couple of weeks later the same patient developed a slight cough and sent for me. The temperature was, I remember, above 101° F., but not as high as 102° F. There was considerable cough, with slight expectoration of a watery mucus, and at the left base, in a small patch, crepitant râles were heard. Instructions were given to have a fresh specimen of sputum ready for me the following morning for examination, but on my arrival I found that the cough, expectoration, and the râles at the left base were all gone, and that the temperature was normal. Considering the fact of the previous œdema and the sudden onset, and as sudden disappearance of the lung condition, the point at once suggested itself that here we might have had a localised pulmonary œdema. It would be well, therefore, to carefully observe such cases where localised œdema has been noted for a like temporary lesion in the lungs or other organs.

Etiology.—Like Dr. Dalgetty, I suspected *filaria* and searched a good many blood films in various cases, but I cannot say that I have yet searched enough films to be able to decide as to the invariable absence of filaria, which I shall continue to suspect as the cause of the disease, until proof to the contrary is forthcoming. It may be noted, however, that elephantiasis and lymph scrotum are very rare in Siam. The difficulty has been that most of the sufferers have been members of the nobility, and so one could not make unlimited blood films in these circumstances.

In one case the *plasmodium of æstivo-autumnal fever* was detected in the blood. In connection with this fact, a suggestive case may be mentioned of œdema of the ocular and palpebral conjunctiva of one eye in a case seen by me in Singapore some years ago.

The following are brief notes of the case:—

A wealthy half-caste, fleeing from Manilla at the outbreak of the rebellion against the Spanish, stopped at Singapore for a few weeks, where he had a slight attack of ague and fever.

The morning following the ague, I noticed œdema of the ocular and palpebral conjunctiva of the left eye.

The patient stated that this was a common sequela of an attack of ague with him.

The following day the œdema was practically gone.

Chill may induce the œdema, as in a case well known to me where cold baths always bring out localised œdema of the forearms.

Traumatism of any kind or poisoning from the bite of insects plays no part in the etiology, I am thoroughly convinced. As to *nationality* of cases, Dr. Posnet states that in Brazil it is comparatively common amongst Englishmen, but rare in Portuguese or Brazilians.

Dr. Dalgetty makes no mention of the nationality. I have seen it amongst Siamese almost entirely, but one well marked example was a Chinaman in Singapore. I have never seen it amongst Europeans, nor have I heard of its attacking them. Dr. Dalgetty's Indian cases are more like the type which prevails in Siam.

I have never seen such severe cases with constitutional symptoms as Dr. Posnet has noted in Brazil, nor have I noted more than one local swelling at a time on the same individual. However, the affection is as much a puzzle to me as to my colleagues who have already written upon it, and I have long noted the disease as one requiring careful investigation, especially as no mention can be found in the usual text-books on Tropical Diseases.

NOTES ON YAWS IN TRINIDAD, W.I.

By R. C. Bennett, M.B., C.M.
Government Medical Officer.

Yaws has assumed such proportions in Trinidad, and has become so serious a nuisance, that special attention has been given to it by the local Government, and has induced me to give it special study and submit a few notes.

Nomenclature.

Many names have been given to this disease by different writers in different parts of the world, which has led to some confusion, and opens some reasonable doubt as to whether the affection described under various names in the affected areas are pathologically identical.

There is the Brazilian frambœsia, or boubas, of Professor Breda, and the Polypapilloma tropicum of Charlonis and others.

The natives of different countries have their local names, and even the people of one colony have many names for the same disease according to its site, its stage, or the condition of parts affected.

Frambœsia has been a generally accepted term, but it would be better to adhere to the simple name, yaws.

Its History in Trinidad.

Trinidad is an island situated about 10° north of the equator, between 61° and 62° west longitude in southern part of Carribean Sea. It is separated from Venezuelan coast of South America by Gulf of Paria, and is the second largest of British West Indies. It is the most prosperous, and has been under British rule for a century. Population over a quarter of a million—mixed—one-third being Indians imported from India and born in the colony (Collins). Average temperature about 80°-85°, and there are two seasons, the dry and the wet. The atmosphere be-

comes saturated with moisture during many months of the year.

Yaws was introduced into Trinidad by slaves from Africa, though there is no record of importation *direct* from that country. I have been able to obtain positive information of the existence of cases as far back as 1807. The disease must, however, have been in existence before that date. Finding a suitable *nidus*, and favourable conditions for its growth, it has steadily increased since then, and has assumed such proportions as to have become a social nuisance and a menace to the community.

In 1783 the records of the Cabildo show that there were only 310 slaves in the colony, but when the English captured the island that number had increased to 20,464. This was owing to the large number of immigrants after the publication of the famous Cedula of population issued by the King of Spain permitting foreigners to settle in Trinidad. These brought their slaves with them from the other islands, but how great a proportion were African by birth it is impossible to say. It should be added that subsequent to emancipation many immigrants were introduced from Sierra Leone and other parts of the west coast of Africa, and a number of liberated Africans taken from slavers were also brought here, the total number of free African labourers introduced between 1843 and 1861 amounting to 6,375.

"The attempts at isolation hitherto made, even in such prosperous places as Trinidad, have never been on a sufficient scale to arrest the spread of the contagion, still less to ensure its extinction" (*Numa Rat*). There are more cases of yaws in Trinidad now than ever; the provisions for dealing with the affection are, however, better than hitherto, but cannot and will not permanently wipe out the disease.

When Dr. Nicholls visited this colony in 1891, preparatory to his valuable official report on yaws, the Local Government was advised that there was no necessity for an inquiry, on the ground that there were very few cases in the colony; therefore Dr. Nicholls had little to say about yaws in Trinidad. No returns are available giving the number of cases at that period. Five years later (1896) yaws was so prevalent in the island as to necessitate the introduction of a Yaws Ordinance, and in 1899 (last year) the number of cases officially reported as *under treatment* were 2,808. Not one-half of these cases were cured during that year.

It has been stated that yaws is a disease confined to the African race. This is not so. It is common here amongst the coolies, both those imported from India as well as those born in the island. I have never seen a case in a white subject; and this might be explained by the fact that they live in a better and cleaner atmosphere, and under less unfavourable conditions than the ordinary victims of yaws.

Nature of Disease—Its Cause.

(*a*) *Primary.*—No specific micro-organism has been discovered in the tissues or by cultures, and one might be inclined to conclude from the clinical description of the cases of Brazilian frambœsia or buobas, by Professor Achilles Breda, and in which he announces the discovery of a bacillus, that the affection there differs in some marked manner to the disease here. One does not gather that the Professor made any attempts at growing the bacillus.

(*b*) *Maintaining.*—Apart from micro-organisms yaws may be considered as exclusively a dirt disease, propagated and maintained by dirt and occurring in dirty people of dirty habits and under dirty circumstances and surroundings. Yaws is hardly ever seen in persons of cleanly habits. In notes on treatment reference to the dwellings of the subjects of yaws will be made.

Diagnosis and Symptoms.

The diagnosis, in an established case of yaws, presents no difficulty, and should seldom or never be confused with other affections of the skin.

Dr. Rat, in his Essay on Frambœsia (1891) states that invasion begins at the seat of inoculation as a primary sore "as characteristic of the disease as the chancre is of syphilis." I have not noted this in any of my cases, though I have to admit that I seldom see them in very early stages. The allusion to syphilis in connection with yaws seems unfortunate.

In the hundreds of cases of yaws seen in Trinidad I have never met a single case in which the lesion was confined to the mucous membrane, though the junction of skin and mucous membrane is a common site. The coloured plates which accompany Professor Breda's report on Brazilian Frambœsia show the glottis, epiglottis, base of tongue, palate, soft palate, uvula, mucous membrane over arytenoid cartilages, &c., to be affected by papillary growths and ulcerations. It is not asserted that in cases here the mucous membrane does not become affected—only that I have not seen a case.

The constitutional symptoms of yaws are in the majority of cases almost *nil*.

Relation of Yaws to other Diseases.

(*a*) *Leprosy.*—Yaws occurs in leprous subjects as it does in persons suffering from other lesions. A case came under review in which it was evident that yaws, leprosy, and tertiary syphilis existed in the same patient, and as distinct diseases. Has yaws any relation to, or connection with, leprosy? The answer should be in the negative. Some writers assert the contrary, and it has been stated that "Leprosy (Arabian and Grecian) are only varieties of one disease which has originally sprung from yaws" (New Sydenham Society, 1897). Similar statements have been made founded on evidence taken from the Old Testament.

There is evidence of yaws as far back as the tenth (10th) century. Leprosy existed anterior to that period; and unless there is positive evidence to the contrary, the suggestion that yaws had given birth to leprosy must be abandoned.

Taking the most elastic view of the theory (or the fact) that diseases are modified by time, circumstances, climate, &c., there are very few, if any, clinical features in common to warrant any relationship between the two diseases. Nor is there any pathological relationship. Where are in yaws cases, amongst other things, the trophic-nerve lesions with their ghastly and destructive train of symptoms and changes in tissues, bones, &c.? Where are the constitutional symptoms, where the hereditary transmissions, &c.?

Leprosy is due to a specific bacillus invariably present in the tissues and serum from tubercles. No specific bacillus of yaws has been discussed. This should suffice to settle doubts. A recent suggestion, but a doubtful one, is that leprosy arises *per se*. There is no doubt, however, that yaws is a disease *sui generis* — contagious and chronic, but curable. Leprosy is incurable (and its contagion is denied by some and strongly asserted by others). Not so with yaws.

(*b*) *Syphilis.*—Has yaws any connection or relation to syphilis? With deference to others I submit not, though the question should be finally disposed of.

Syphilis is a well known specific and constitutional affection, with a distinct history and usually pronounced clinical features and sequelæ. Where are those in the thousands of cases of yaws that have been treated in the West Indies? In yaws cases there is no history of hereditary syphilis, and children affected are as a rule otherwise healthy and fairly developed, in spite of the affection. Some cases are debilitated from other causes or from the irritation and exhaustion of a protracted and chronic eruption of yaws.

If the affection was of a hereditary syphilitic nature it would be reasonable to ask why infants show no signs of yaws (or syphilis) at birth, and why no symptoms develope until months or years after birth, and why when after such protracted periods in the large majority of cases only local manifestations of yaws with entire absence of constitutional symptoms manifest themselves? There is not a single case on record where a child has been born with yaws.

If the large number of cases in this colony (I have just at present under treatment 140 cases) are of hereditary syphilitic origin, a very large number of adult parents must also be syphilitic. Any such suggestion would be an outrage on the community (*vide* return of cases for one year—about 3,000; pop. 250,000).

To connect yaws with syphilis is to hamper the study of the former, and to direct treatment into channels more in keeping with the latter disease.

Immunity from Second Attacks.

It has been stated that a first attack confers immunity. Some deny this, and add that there is a long interval between the attacks.

I discharged a boy perfectly clean; a few weeks after he returned with a fresh crop of yaws. It is more likely that this was a retarded outbreak of the original infection rather than a case of re-infection. Watch many cases of yaws carefully, and it will be seen that on the eve of discharging them "cured and clean" a fresh crop or eruption suddenly appears. These are not cases of reinfection.

I discharged, cured, a boy (aged 7 years) on March 16, who had been under treatment for some time. He returned on May 25, with marked indications of yaws on arms, feet, &c. This looks like a case of re-infection, but need not necessarily be so. The question of immunity from second attacks requires more substantial proof, though my experience leads me to say that re-infection does occur, and that one attack may have a temporary, but not a permanent immunising effect.

Contagion.

(*a*) By direct contact. Yaws is contagious by direct contact. The negroes on the sugar plantations at the time of slavery knew this, and practised inoculation for motives of their own. All cases were isolated and treated in the plantations, which meant a cessation from work. Mothers inoculated their infants so as to obtain an excuse for remaining at home to cure them.

An abrasion, ulcer or sore, or some solution of the continuity of the superficial tissues, seems an absolutely necessary condition in contagion. The interesting inoculation experiments in man by Dr. M. Charlonis, in which he produced yaws by direct inoculation, has settled the question of contagion.

This writer also states that it appeared to him so remarkable that a child should suffer from the disease upon the lip without infecting its brothers and sisters who eat and drink out of the same vessels, and sleep with the patient in the same room. It would be remarkable in one way, *i.e.*, if the brothers and sisters suffered from abrasions or some other solution of continuity in the skin. Not otherwise.

This writer, in his interesting contribution on Polypapilloma tropicum (frambœsia), also adds that "he has only very seldom seen two children of the same family afflicted with yaws." It is by no means uncommon here to see almost every child in a household affected.

It is almost impossible to get data as to the time of infection in a case of yaws, especially as the period of incubation varies so. A subject infected may give no local indications of the disease for months. The period of incubation may extend over three months.

Rodschied and Brunce state that frambœsia may be preceded by a prodromal stage lasting for weeks, during which there is loss of appetite, an unpleasant taste in the mouth, and according to Ferrier, vomiting. Charlonis also makes mention of a period of fever with attendant gastric disturbances. These are all such common symptoms in tropical ailments that it becomes impossible to seriously take them as being premonitory of an invasion of yaws. Even in the very early stage of the eruption, in some cases a correct diagnosis might be hazardous.

(*b*) By indirect contact: through the agency of a fly or other insect.

Flies are supposed—and it has been so stated—to carry the contagion, and in Trinidad, one hears of a "yaws fly"—but never sees it, and its identity remains concealed. I doubt not the possibility or probability of the yaws virus (whatever it may be) being so conveyed, but when the true nature and cause of a disease is not accurately known any theory or statement under the face of the sun is a possibility or a probability—or both. There is positively no evidence here that yaws is conveyed from one person to another through the agency of a fly.

Inoculation.

(*a*) In man. This has already been demonstrated.

(*b*) In birds. In this colony an affection occurs in domestic fowls at certain periods, affecting the head, which becomes covered with thick, hard crusts. It occurs epidemically. From its resemblance to frambœsia in the human being the disease is called locally

"yaws." It has no pathological relationship to human yaws. (Nicholls.)

On March 20, I inoculated a half-grown fowl with fresh yaws tubercle, taken from a boy's foot, after it had been denuded of its crusts and rendered aseptic.

A deep incision, under aseptic precautions, was made into muscles of the bird, portions of tubercle introduced and wound closed with horse-hair sutures.

The bird was caged—its temperature was 107° F.; the bird showed no bad symptoms, the wound healed, it was kept under observation for three months, but showed no signs or symptoms of any affection whatever.

On the same date a Barbary dove was also inoculated, its temperature 107·4°. Portions of same tubercle under similar precautions were introduced, not into the muscles, but subcutaneously, and wound sutured.

The bird drooped and died one week after. *Post mortem*, the wound had practically healed. The yaws tubercle had been absorbed, no skin affection, lungs soft, and at parts caseous. Similar deposits in intestines. (Preserved for future examination.)

Pathology and Histology of Yaws.

The pathology of yaws has been well described, and in a short paper like this no useful purpose could be served by reviewing it.

I made microtomic sections of yaws granulomata from different subjects, and single, double and triple stains brought out clearly the tissue changes so clearly illustrated in different works.

Bacteriology of Yaws.

The bacteriological study of yaws has not up to now been concluded, though some work has been done in that direction.

The enormous benefit that has been obtained by the bacteriological study of diseases in modern times induces one to apply it with fair amount of hope in searching for a specific cause in this very obstinate disease.

Dr. Nicholls considers it "clear from a consideration of the attributes of the disease that it is due to a microbe." This microbe he speaks of as the "yaws micrococcus" which he found in "abundance in lymphatic glands examined" and "in other organs of the body." He also found "a micrococcus invariably in the secretion of the granulomata."

He also "succeeded in cultivating micrococci from dust swept from the floor of the Yaws Hospital."

I do not think there are sufficient grounds, and in the absence of further bacteriological work, to accept Dr. Nicholl's "yaws micrococcus" as being *the cause* of the disease. Cocci are pretty universally distributed, and must always be present in any morbid secretion of yaws or other local affection exposed to air, dirt, &c., and undergoing inflammatory changes. The presence of these micro-organisms in lymphatic glands, &c., were, I should think, secondary.

Professor Achilles Breda, in his contribution to the "Clinical and Bacteriological Study of the Brazilian Frambœsia," or "Buobas," describes a bacillus discovered by him. He used a high magnification, $\frac{1}{18}$, homogenous immersion, and describes the bacilli as measuring 0·3—0·45 in. in length and 0·08 in. in breadth. He found them in tissues and sections taken from affected skin beneath the capillary layer; and again, beneath the peripheral capillary layer. He also found them in the stroma of the uvula, and soft palate. Some were seen lying within a capillary vessel, and others in proximity to it.

I have already stated that I do not think that the Brazilian frambœsia of Professor Breda is identical with the yaws of Trinidad. There are marked clinical differences. I made the most careful examination of dozens of stained sections with $\frac{1}{12}$ oil immersion lens, but failed to find a bacillus, and I used some of the identical methods in staining as did Professor Breda, and in addition many more. The single, double, and triple staining gave negative results. Here, again, there is a difference: in the Brazilian frambœsia a baccillus; in Trinidad yaws, none.

Scrapings from surface of yaws granulomata preserved in glycerine and obtained from different patients in different parts of the colony I dealt with bacteriologically. To sterilised broth media some of these scrapings were added and incubated at 35° C. In twenty-four hours the growth, yellowish and turbid, was examined and found to be cocci, diplococci, tetracocci, and staphylococci.

Subcultures on gelatine and nutrient agar-agar were made from this, and colonies, of staphylococci grew, and so did colonies of a bacillus which on further examination turned out to be the Bacillus subtilis. From both the scrapings, from different patients, obtained at separate districts of the island, the Bacillus subtilis (which is a non-pathogenic organism) was grown. A colony of streptrococci was also grown.[1]

Conditions favouring spread of yaws here are three:—

(1) The unclean habits of those affected and an utter indifference to the disease. The negroes and coolies affected are well aware of the contagious nature of the disease, but they take no precautions to separate the affected from the non-affected children. Living, as most of them do, in outlying and only partially settled districts, they live and grow ignorant of a single law of hygiene, and entrust the regulating of all sanitary laws to a merciful tropical climate.

Their water supply in some districts is insufficient and in many instances bad. The houses or huts are badly and roughly constructed. Their sleeping compartments are dark and stink, every particle of light is excluded in many cases. Some of these huts, more so amongst the coolies, are not floored, and to enter, one has to double up, the roof coming to within a few feet off the ground. In these dwellings children are born and they live and grow there in an atmosphere and under conditions most favourable to the maintenance of yaws.

(2) Local conditions and the absence or non-enforcement of any practical sanitary measures in the out-lying districts.

(3) The non-isolation of all affected persons.

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[1] The histological and bacteriological work was done at the London School of Tropical Medicine during my residence there.

MALARIAL ULCERS IN BRITISH CENTRAL AFRICA.

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Bale and Danielsson, Ltd., London.

Treatment.

(*a*) *Hygienic*: (*b*) *Constitutional*; (*c*) *Local.*

The treatment adopted by the local Government is as good as it can be under *present* circumstances, but it will *never succeed* in entirely wiping out this troublesome disease. It consists of a Central Hospital and District Yaws Dispensaries. These last work fairly well, but unfortunately are helpless at isolation and have some difficulty in regulating attendance—though all persons affected with yaws obtain treatment and medicines free of any cost.

Though many cases are cured, fresh ones keep cropping up. The foci of infection remain.

The cause is not removed, which is the first principle of treatment of diseases. This is a serious drawback to the outdoor or dispensing treatment of yaws, which should be gradually abandoned in time, in favour of more elaborate Central Yaws Hospitals.

(*a*) *Hygienic.*—Not easily carried out owing to local conditions, but is sound in principle and demands consideration. With better supply of water, and the official regulating of dwellings, and some instruction on the A, B, C, of sanitary measures, the natives will in time learn that cleanliness is a good preventive measure. No one is more alive to his interest than the native after being impressed morally and legally.

Taking the number of yaws cases in Trinidad (which includes Tobago) during the last year (1899), as three thousand (3,000), one has to candidly admit that we had 3,000 foci of infection amongst the inhabitants.

A very large majority of cases occur in children, and allowing a large margin in this direction, I fear it must also be admitted that at least 2,000 infected children must have been prevented (legally) from attending the public and Government schools. This is a serious matter. Yaws has spread and increased in this colony to such enormous proportions as to have become a social nuisance and a scourge.

The Surgeon-General of the colony, who has interested himself much in the matter, stated in his last official report (1899), that "returns show a great increase of the disease in most parts of the colony."

(*b*) *Constitutional.*—The treatment of yaws by drugs is somewhat empirical, as it must be when the specific cause and the true nature of the disease is not correctly known. Especially so when a most distinguished member of the profession considers it "syphilitic" and nothing else. *Mercury*, though recommended, might be permanently discarded.

Iodide of Potassium is useful in some cases, though I have seen hardly any beneficial results in many cases after a protracted administration of this salt. It semed in many cases to have no effect whatever, and did not prevent the eruption of successive crops of yaws.

Arsenic gives fairly good results. *Iron* also is useful, possibly in an indirect manner. Large doses of tincture of steel, especially in children, has given excellent results.

Guaiacal and thyroid-gland extract have been pressed into the service and advocated in the treatment of yaws. I have no experience of the former, and cannot see on what grounds any good results can be expected by the administration of the latter.

(*c*) *Local.*—The local treatment of yaws has, I fear, been pushed aside in favour of internal treatment, and has not received, perhaps, as much attention as it might. The treatment is somewhat heroic, but is justified by the results, and can only be applied in suitable cases. It consists in the use of the knife and the application of strong nitric or carbolic acids and caustics.

Inspect any collected number of yaws cases in or out of a hospital. Amongst them you will see many cases with large, very prominent granulomata; inquire, and you will find that these cases have been under treatment (internal) for many months, and that though the patient has improved in general health, with no fresh outbreaks of yaws, the large granulomata have given no indications of contraction, softening, or absorption. Further internal treatment in these cases is useless. I regard these granulomata as neoplasms, new growths, made up of proliferated and hypertrophied tissues, and as secondary results (in a manner) of the primary affection. Remove them with the knife, and your patient leaves the hospital quite clean in a few weeks. Continue to treat him medicinally, and he remains in hospital many months, perhaps over a year.

The method is simple. Select a prominent granuloma, wash with antiseptic, and, if desirable, apply sol. cocaine to relieve pain; with sharp knife slice it clean away at its base, and apply swab of carbolic or nitric acid to cut surface. Some cases do not permit of the knife; these, after being ringed with vaseline, are swabbed with nitric or carbolic acids. Notes of two cases are given:—

(1) W. A., aged 4 years, had been under treatment for over three months, up to February 23. On this date two enormous granulomata on right foot, one on left, and one on hand, were sliced off, and HNO_3 applied. March 2, change marvellous; March 9, clean, healthy surfaces. Some nitrate of silver applied to foot only. March 23, discharged quite well. Internal treatment was discontinued from date of operation.

(2) W. P., aged 17 years, two large granulomata involving greater part of cheeks. Had been under treatment up to March 20 for four months, during which time he had taken freely and steadily arsenic, pot. iodid., guaiacol—ointments had been applied to face. Results absolutely *nil*. I hesitated applying heroic measures to the face, but he had become depressed at the disfigured and loathsome appearance of his face. On above date the growths were carefully ringed with vaseline and strong nitric acid applied. March 27, clean surface, to which pencil of nitrate of silver applied, which was repeated on subsequent occasions. May 1, discharged cured; skin of affected part slightly darker than the normal. No disfigurement owing to treatment. He had been over sixteen weeks under continuous medicinal and internal treatment, and had not improved in the least; under the local treatment he was discharged quite clean under six weeks.

Concluding Remarks.

(1) Yaws is a disease *sui generis*.
(2) Contagious, and on the increase.

(3) Due possibly to specific micro-organism not yet established.

(4) No connection with or relationship to leprosy or syphilis.

(5) Treatment, hygienic and general. No specific.

(6) Local treatment in proper cases important.

(7) Isolation in central yaws hospitals only means of dealing (with serious view of extermination) with yaws, a disease that has been endemic in this island for over a century, and is on the increase, very seriously affecting a large section of the labouring community.

SOME NOTES ON THE DISEASES OF MASHONALAND.

By Charles Todd, M.D.

In view of the rapidly-increasing importance of Rhodesia and the scarcity of literature dealing with its diseases, the following observations may be of interest. They cover a period of two years, and were made when acting as medical officer to the contractors of the Beira and Mashonaland railways.

The district under observation extended from Mandigos, in the Mozambique Company's territory, seventy-two miles to the Chartered Company's boundary, and from there for about a hundred and seventy miles through Mashonaland to Salisbury; but as at the time there were very few railway *employés* located between Mandigos and Umtali, the following remarks refer almost entirely to the Mashonaland portion of the section where a large number of men were employed in the construction of the Mashonaland railway.

The country through which the line passes from Mandigos to Salisbury is very hilly, and in places mountainous, though on the high veldt, near Salisbury, there are stretches of open rolling country. The formation is almost entirely of granite.

Mandigos is about 2,000 feet above the sea level, and from there the line rises almost continuously to an elevation of almost 5,000 feet at Salisbury. The water sheds are followed as much as possible so as to avoid the innumerable small streams which course down the hill sides during the wet season, but in several places, especially near Salisbury, the line crosses small swampy areas consisting of black alluvial soil lying in large shallow pans in the granite, and so inefficiently drained and water-logged. These areas formed fruitful sources of malaria during the construction of the line.

This part of Mashonaland has a definite wet season, coinciding with the season of high temperature, but it is not so definitely limited as it is nearer the coast.

The respective years vary very greatly as regards rainfall, and meteorological observations extending back for any time are not yet obtainable, so that it is impossible to make any definite statements as to the average rainfall. Mr. Reischack, curator of the Umtali Park, has, however, furnished me with the records taken in Umtali Park during the latter half of 1898 and the whole of 1899, which give a good idea of the climatological conditions.

Mosquitoes, though very troublesome in the low country, and, indeed, up to the boundary, are only seen—between Umtali and Salisbury—in the wet season, and are not numerous then.

I have never seen a member of the *Anopheles* group on the Chartered Company's side of the boundary, though it is quite possible they would be found if systematically looked for.

The appearance of mosquitoes in Umtali, both in 1899 and 1900, was first noticed just before the rise of the malaria curve, and though these were all members of the *Culex* group, it is not improbable that their appearance coincided with that of specimens of *Anopheles* which escaped observation.

Malaria.—This was the most important disease affecting the staff, the number of cases varying from about 5 per cent. per month during the healthy season to as much as 30 per cent. per month during the fever season. This is the number of cases which received medical treatment, and there were, doubtless, many mild cases which did not come under observation. As in the majority of cases it was impossible to decide whether the attack was a re-infection or merely a relapse, attacks of malaria occurring after a period of over six weeks from a previous attack were counted, for purposes of enumeration, as separate attacks.

Two types of malaria are met with, viz.:—

(1) A simple or double tertian due to the ordinary tertian parasite, which chiefly occurs during the latter part of the dry season when the malaria curve is low, and

(2) A remittent form prevailing during, and for some months after, the rainy season. This latter form is due to a parasite which, in its early stages, is seen in the red blood-corpuscles as a small, clear sphere or ovoid, sometimes unpigmented, but usually showing one or two nucleolar-like granules. It stains badly with aniline dyes, and the riper forms are not as a rule to be found in the peripheral circulation. I did not have an opportunity of examining the spleen or bone marrow in an acute case, so that it was impossible to follow the further development of the parasite.

Considering the close resemblance of the parasite to that of the æstivo-autumnal form, one is much struck by the absence of crescentic forms in the blood, and although these were looked for both at varying periods of time after the attack and in many individuals, I was quite unable to demonstrate them in any of the cases.

This remittent form, though it gave rise to severe illness, was not as a rule malignant, and was most amenable to treatment with quinine; but somewhat large doses were necessary, whereas the tertian form yielded to quite small doses.

In the severe remittents it is most important that the administration of quinine should be commenced as early as possible, and in serious cases it is often advisable to administer the acid hydrochloride hypodermically, as by this means its rapid absorption is ensured.

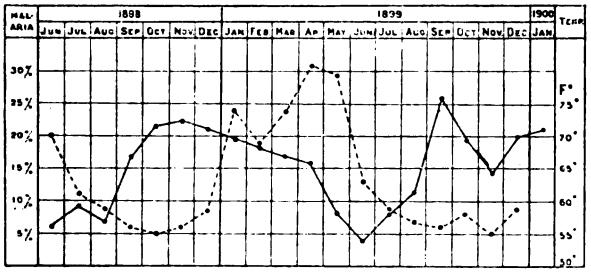
In the more severe cases hæmorrhages were not uncommon, and in one or two cases were very severe. In one case hæmorrhages occurred from the nose,

stomach, lungs, bladder, and rectum, and the patient lost a large amount of blood, becoming intensely anæmic. Under the influence of a mixture of calcium chloride and liquor ferri perchloride, however, these hæmorrhages ceased, quinine being, of course, administered at the same time.

Calcium chloride, when given with the perchloride of iron, appears to be especially useful in the hæmorrhages of malaria, some of which are very troublesome, *e.g.*, the *post-partum* losses of women who are subjects of malaria.

Appended is given a table showing the incidence of

MALARIA AND MEAN TEMPERATURE (UMTALI, RHODESIA).



- - - - Malaria. Percentage of staff affected.
——— Mean Temperature. (Umtali.)

RAINFALL—UMTALI (IN INCHES).

1898		1898		1899		1899	
June ..	2·45	December	—	May ..	0·51	November ..	7·47
July	0·04	1899		June ..	0·38	December ..	7·36
August ..	3·00	January ..	—	July ..	0·31	1900	
September ..	1·05	February	21·15	August ..	0·01	January ..	13·26
October ..	2·14	March ..	1·14	September	0·00		
November ..	3·05	April ..	1·62	October ..	1·72		

malaria over a period of twenty months, for which trustworthy numbers were available, together with the mean temperature and rainfall. This table shows the effect of heavy and sudden rainfall in checking the incidence of malaria. In February, 1899, when the malaria curve was very high and still rising, some very heavy rains occurred, over four inches falling in one night and twenty-one inches during the month. This caused a sharp temporary drop in the malaria curve, which, however, rose steadily during the two months following. Sudden heavy rains appear constantly to temporarily diminish the amount of malaria, this possibly being due to the rain either drowning the mosquitoes or washing out the pools containing their larvæ.

The figures given would appear to show that Mashonaland is a most malarial country, but it must be remembered that the class of men on whom these data are based were leading a life of exposure, and at the time were turning over new soil in the construction of the railway. There was also a great deal of bridge work involving the digging of foundations, and much work in the neighbourhood of rivers and streams, and it was noticed that men engaged in this work, especially in certain places, were particularly liable to malaria.

When these numbers are compared with similar ones relating to persons living in the town of Umtali and Salisbury, and whose work did not necessitate exposure, the results are most striking. This is especially seen in the case of women and children living in the towns, who very rarely contract malaria.

An interesting point in the ætiology of malaria is furnished by the experience of old hunters in the low country, who, as a rule, agree that a mosquito net will keep off the fever when camping out on the plats. This they attribute to its keeping off the night mists, the idea that the mosquito may be a source of danger not appearing to occur to them.

The *rôle* of the mosquito in the transmission of malaria being now generally acknowledged, it appears not impossible that other parasitic insects may play a similar part.

When living in Umtali, I became the possessor of a family of five kittens, who were the means of introducing into the house an invasion of fleas from the neighbouring sandy soil. The discomfort became so great that the kittens had to be dispensed with. About eight or ten days after being severely bitten I developed a sharp attack of malaria. This was, of course, no proof that the flea is able to act as a carrier of malaria, for, as I had previously suffered from malaria, it was impossible to say whether the attack was a re-infection or a relapse; but it was suggestive of the possibility of such a means of transmission. Unfortunately at the time I was too busy to give the matter further attention.

An opportunity of testing the prophylactic value of quinine occurred during the wet season of 1900, when a party of nine or ten locomotive engineers, fitters, &c., was sent down to Amatongas, in the low country, and remained at work there for about a month. These men took quinine regularly during their stay in Amatongas, each man taking ten grains a day for the whole period. It is interesting to note that every man of the party had an attack of fever, either during his stay in Amatongas or within a week of his return to Umtali.

The natives living in the district possess a very high degree of immunity against malaria, their attacks being infrequent and of a very mild type. Natives coming into the district from other parts are more liable to malaria than those native to the district.

During the two years (May, 1898, to May, 1900) rather over seven hundred cases of malaria in whites were treated—exclusive of blackwater fever—with two deaths, one of malarial coma, and the other of post-malarial cachexia in an elderly man.

The fact that the contractors provided excellent accommodation for the men, who were, wherever it was possible, located in wood-lined iron houses well elevated on piles, no doubt had a great influence on the fever mortality and the general health. Quinine was freely supplied to the men, who were instructed to continue taking it for at least a fortnight after an attack, and if possible for a month.

Blackwater fever was not common. In seven cases in which I had an opportunity of examining the blood, parasites were found in three. These were indistinguishable from those of the remittent form of malaria mentioned above.

Apropos of Koch's views one case was interesting, as the patient had never taken quinine until after the onset of the hæmoglobinuria. He was most definite in his statements when questioned on this point.

Quinine appears to have absolutely no effect on the disease, and after giving it a fair trial its use was discontinued. Large enemas of hot water appear

to do more good than any other form of treatment, checking vomiting and relieving the thirst, besides acting, presumably, reflexly as a renal stimulant.

The relief effected in this way is sometimes most striking.

Beri-beri is not common amongst whites, but natives are not infrequently attacked. A form of peripheral neuritis was noticed as attacking especially Inhambane boys. This occurred both in the paraplegic and dropsical forms and was indistinguishable from beri-beri, but was never accompanied by anæsthesia. Death commonly occurred from cardiac paresis.

Dysentery is common at certain seasons, but is as a rule of a very mild type and most amenable to treatment. Monsonia ovata was tried and in some cases gave excellent results, but in others appeared to have no action. Sodium sulphate was found by far the most useful drug, especially when given frequently and with a large amount of fluid.

A form of continued fever resembling Malta fever.—A small epidemic of about a dozen cases of continued fever was seen in Umtali. These cases were at first diagnosed as typhoid, but as the cases progressed they were seen to present certain peculiarities which led to their being regarded as probably cases of Malta fever.

The diagnosis was based on the following points :—

(1) The character of the temperature curve, viz., "Intermittent waves of remittent pyrexia."

(2) The long duration of the attacks.

(3) Very profuse sweating occurred in all the cases.

(4) Constipation was the rule—in every case.

(5) The absence of typhoid spots—in all the cases.

(6) The severe rheumatic and neuralgic pains.

As none of the cases ended fatally it was impossible to verify this diagnosis by bacteriological methods, and as no cultures of the micrococcus could be obtained, Widal's test could not be applied.

Curiously enough, about twelve months later, in the course of conversation with one of the patients who had suffered at the beginning of the epidemic, he informed me that shortly before his illness some imported sheep from Malta had been located immediately behind his house. I do not know if there could be any connection between these facts, but they are at any rate interesting.

Malta fever is not described as occurring in South Africa, but it is quite possible that cases of this disease occur amongst the cases classed as "typho-malarial," "camp fever," &c.

Veldt sores.—In the autumn these are very common, more especially amongst men who have been more than a year in the country. At this time in some persons the superficial layers of the skin appear to become soft and sodden, so that abrasions take place with great ease. These abrasions do not heal, but form eczematous ulcers which spread laterally but never attain any great depth.

Exposure to air and light greatly delays the healing process. A change of climate from the high veldt to the coast has a most marked effect, the healing process usually beginning at once on reaching the coast.

Dr. R. T. Michell, in a communication to the *British Medical Journal* (May 1, 1897), suggests that these sores are connected with a scurvy-like condition of the blood. In some instances this may be the case, but I have repeatedly seen them occurring in men who were living well on fresh meat with abundant fresh vegetables, and have, indeed, experienced them myself under similar circumstances. Of local applications, a weak solution of formalin gives perhaps the best results, but has the disadvantages of being painful and of producing a certain amount of pigmentation if applied for any length of time.

Second to change of climate, the shielding of the ulcers from the light and air is undoubtedly the most important factor in the treatment.

Enteric fever occurs sporadically, but fortunately no epidemics were seen. The "pail" system of sewage disposal is used in the towns, and is as a rule remarkably well carried out.

Parasites.—The "jigger," or "mataquinho" as it is called by the Portuguese and Kaffirs, is now disseminated throughout Mashonaland and is a most annoying pest, the naked feet of the Kaffirs forming an excellent means of distributing it broadcast.

Another noxious insect is a fly called by the Zambesi natives the "marooma," and by the coast boys the "effungheri" fly. It is a metallic green coloured fly about the size of a bluebottle, and possesses a telescopic ovipositor with which it deposits its eggs under the skin of the unfortunate victim. The eggs are hatched in this position and a pustule containing a large maggot results. The pustule ultimately ruptures and its occupant escapes.

Odd cases of bilharzia are seen, but they seem to be all imported.

Other diseases.—No cases of scarlet fever, diphtheria, acute rheumatism or malignant disease were observed. Two separate cases of variola and one of morbilli were seen, but these were all introduced from the coast and did not give rise to further cases. Leprosy is rare, and tuberculosis, though occurring to a certain extent amongst the Mashonas, is not common.

Many cases of pertussis were seen in Kaffirs and this disease appears to attack adults in many cases.

One or two cases of varicella occurred in Kaffirs but none in whites.

Bronchitis is exceedingly common both amongst whites and Kaffirs during the cold season. It also appears to occur as a sequela of malaria.

As in many other tropical countries the nature of the food and in many cases over indulgence in alcohol causes a large amount of dyspepsia, dental caries and similar troubles.

PLAGUE.—In Glasgow there has been no fresh case of plague since September 22, and the Plague Hospital is empty. In Hong-Kong there were no cases of plague during the week ending November 10. In India plague is serious in the Mysore city and province, and accounts for almost one-third of the cases in all India.

The slight improvement in the plague returns in districts further north, including Bombay and Calcutta, encourages the more that the fifth epidemic which now threatens may be averted, or at least that the outbreak may be less prevalent than its predecessors.

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THE

Journal of Tropical Medicine

NOVEMBER, 1900.

THE RESTING POSITION OF ANOPHELES.

THE familiar diagram published by Ross illustrating what he, at the time, regarded as the characteristic position assumed by the malaria-bearing mosquito, and which has been reproduced in almost every work and pamphlet on malaria issued during the past two years, is now regarded by several observers to be incorrect.

In the history of every important scientific discovery some details require subsequent modification. It is the penalty we pay to a busy age. Ross and others did not wait to ascertain whether the observations they had made concerning the resting attitude of the Indian and African species of Anopheles held good for all species. We shall see, however, that the point is not one of great practical importance. As a matter of fact we cannot recall any discovery of equal importance and of such extreme complexity as the mosquito phase of the malaria parasite which has required so little modification. The cycle of Ross is the same to-day as when he published it in India, and Manson explained it to us in England in 1898.

Ross's description of the position assumed by the species of Anopheles with which he was personally acquainted, namely *A. costalis* and *A. funestus*, is undoubtedly correct and has been confirmed by many other observers. When attention, however, began to be directed to the European species, it was not found to hold good. We believe Guiteras, who after studying at the London School of Tropical Medicine went to Italy to continue his work, was one of the first to observe that *A. claviger* did not assume the characteristic position described by Ross. Grassi also made the same observation and he has entered fully into the subject in his recent memoir. Sambon and Low, on going to Italy, were also struck by the fact that *A. claviger* assumed a position very like that of Culex. These observers, however, made a further discovery for they found that *A. pseudo-pictus*, another European species, rested in the manner described by Ross. Guiteras has also confirmed this in Havanah.

The position of Anopheles when at rest can therefore no longer be relied upon as a distinguishing feature of the genus. It seems to us, however, a matter of very little moment. It was at most only of value to the layman. For the zoologist and trained medical man there are many other distinguishing characters available. We would urge that for the present it is unnecessary and impolitic to attempt to instruct the general public how to recognise the Anopheles. We are not absolutely sure, for example, whether some of the other genera of Culicidæ may not serve as the definitive host of the malaria parasite. It is true that Ross, Grassi, and others have so far failed to find the parasite developing in Culex, but there still remain other genera to be investigated, the Megarhina and Corethra for example. Again, although the Culex may not harbour

malaria we know it carries Filaria nocturna, and from the recent experiments made by the American army surgeons in Cuba, it may be the source of infection in yellow fever. The course which a medical man practising in a malarious country should pursue is clear. He should preach in season and out of season, and to Europeans and to natives, the necessity for protecting themselves against all mosquitoes. He should teach them to regard these insects as their worst enemies. Although this will suffice for the general public, it should not content the scientific mind, and we would strongly urge medical men to miss no opportunity for studying and contrasting the life-history, anatomy, habits and environment of the various genera of Culicidæ. If this be done some important new facts are sure to be brought to light.

THE ETIOLOGY OF BERI-BERI.

By Dr. A. Van der Scheer.

We are indebted to Dr. Manson, C.M.G., F.R.S., for the following valuable communication :—

Dear Sirs,—The accompanying letter, received from Dr. Van der Scheer, of The Hague, formerly of Java, on the subject of the etiology of beri-beri, is worth publishing. The hypothesis therein outlined should be treated. Dr. Scheer refers to a Commission sent by the London School of Tropical Medicine to Malacca to study beri-beri. No such Commission has been sent. I presume he refers to the laboratory at Kuala Zumpoo, under Dr. Wright's direction, which the Government of the Federated Malay States has recently erected, and to which any properly qualified worker in tropical medicine is promised a hearty welcome. A principal object of this laboratory is the study of beri-beri.

Yours truly,
Patrick Manson.

October 25, 1900.

The Hague.

Dear Dr. Manson,—In *The Indian Medical Gazette* (Batavia, Java), I published in the beginning of this year a new hypothesis on the origin of beri-beri, a disease whose spread may be called, perhaps, the most mysterious among those occurring in tropical countries.

In the references given in different journals the reasons that support my idea are so insufficiently indicated that they certainly will not be able to awake much interest in the subject. Therefore I wish to communicate them shortly to you, and as I have read somewhere that some time ago the London School for Tropical Medicine has sent a Commission to Malacca to study the disease, I hope you will draw the attention of the Commission to it, that it may be tested experimentally. My idea arises from :—

(*a*) My not believing that beri-beri is a disease due to the influence of any exogenous toxin emanating from the soil, or present in one or another kind of food. You yourself were not able to find another explanation for the spread of beri-beri than that it took its origin from damp, emanating from the soil and arising by the influence of unknown germs (first edition of your book). This theory will probably not be the real one, because many examples observed by me do not agree with it; and besides, as there is not known one analogous fact in the spread of other diseases that enable us to support the theory. You yourself have avowed that you give it *faute de mieux*.

As to the influence of food: Regarding this subject many instances are known that prove that among people dwelling in different houses, but taking the same food, one group developes beri-beri whilst the other remains exempt from it. One of the most striking examples is one observed in Batavia. The dwellings of native students in medicine are situated in the neighbourhood of the hospital. Several times there has been a little outbreak of beri-beri among them, notwithstanding that they take their meals in the houses of their families at a good distance from their rooms near the hospital, where beri-beri never occurs. Their own rooms serve only for dwelling and studying.

(*b*) Beri-beri is not contagious.

(*c*) Beri-beri is not a miasmatic disease.

I wish to point out that I have become convinced of the truth of this theory during my residence at the hospital in Batavia. It is a collection of large, spacious buildings, separated from each other at a distance of several yards, and occupied by no more than fifty to seventy patients in each. The floors of the "pavilions" are cemented. The water is furnished by artesian wells, the food is excellent, and real miasmatic diseases, as cholera, typhoid, dysentery, never occur in it, although patients suffering from these are often treated within its walls.

Beri-beri occurs there in a severe fashion; the disease seems to laugh at all hygienic measures to bridle its spread. And so we see it everywhere. It appears in hospitals, barracks, &c., which are apparently under the best hygienic conditions; it disappears when the patients attacked (natives) leave the spot and are dwelling in their private houses under conditions much worse than the former.

Whilst the already named miasmatic diseases increase in general, when troops occupy war camps, where water supply, food and bedding are much inferior to those in permanent buildings, and where they are exposed to the influences of bad weather, beri-beri disappears at these times. On the contrary, those people are mostly attacked by it who remain a long time, especially during the greater part of the day, within the infected walls. It is often observed that they are much less frequently attacked when they are out of doors during the day time, although they return within during the night for sleeping. Therefore, hospitals, police-cells and vessels are often notorious centres of endemics. As to the vessels, it is observed that in many cases beri-beri does not

break out in the beginning of a voyage, but usually after several months.

Now it is true that we try to avoid the difficulty in explaining this fact by supposing that beri-beri has a long incubation time. But we know also of many cases in which the incubation is very short, and beri-beri, being an infectious disease, would thus be a strange example of a disease whose time of incubation could differ within the widest limits.

When we see now that beri-beri has neither the features of a miasmatic disease, nor those of a contagious, nor that its spread can be declared by the direct influence of damp or food, we may suppose that it is due to parasites, which are spread by insects (or other animals) that are living within the houses or vessels; the insects would thus form the link that connects the parasites in the human body with those in the surrounding world.

In this way the *Blatta orientalis* seems to me to be very suspicious. Of course it may be also another species of the same family. This animal dwells in all parts of the tropics to which beri-beri is limited (I am not so sure that the epidemic observed in Dublin was real beri-beri). It dwells in our houses and on the ships, and I know one example, that of a ship, the crew of which suffered from beri-beri in an uncommonly high degree, infested by those insects in a way that rendered living almost unbearable.

According to my idea beri-beri must be caused by a parasite (amœba or something else) that lives in the intestinal tube and forms a toxin which causes degeneration of nerves. A part of the life-cycle of the parasite may take place in the body of a blatta species (in its intestines, kidneys, or lymph glands), and the spread of the disease to them would be possible, when:—

(*a*) There are patients suffering from beri-beri.

(*b*) When the blatta species is present.

(*c*) When it is possible that the blatta eats human fæces.

(*d*) When man gets infected by blatta-excrements (fæces, urine, &c.).

When I recall in my mind the epidemics or endemics of beri-beri observed by me, they all, *without exception*, can be explained on this hypothesis. It must be more than an accident that in different hospitals, where convalescents from beri-beri are gathered, and that enjoy always a striking immunity, agree with each other on one thing, that the fæces were not collected in barrels, but were deposited immediately into running water at a good distance from the buildings.

As to the vessels and steamers, it is true, that also here the deposition of fæces takes place into the sea, but the waste pipes in general are directed in a way that infection of blatta may be considered to occur very easily.

I hope these remarks will be the means of exciting interest in this subject, and perhaps direct your attention to the hypothesis. If so, I hope you will recommend the outlines of such a research to the Commission above quoted. As I am not able to take part in it myself, I have thought publication may be the only way by which researches in the direction pointed out by me will have a chance to be undertaken. When, therefore, the Malacca Commission has already finished its work, I hope you will publish an account on this subject in THE JOURNAL OF TROPICAL MEDICINE, and point out some of the parts that appear to you worthy of attention.

You may consider the contents of this letter as a brief extract of my original publication in the *Geneeskundig Tydschrift van Ned. Indie, dl.* 40, April 1, 1900.—Hoping you will excuse my bad English,

I remain,
Yours very sincerely,
A. VAN DER SCHEER.

Address:
Dr. A. Van der Scheer,
The Hague.

RHEUMATISM AS A COMPLICATION OF DYSENTERY.

I RECENTLY had a case of dysentery in which on the third day a rheumatic-like inflammation of both knee joints appeared, and at the same time the dysentery suddenly ceased in such a manner as to attract attention. On consulting the most recent French work on the *Maladies des Pays Chauds*, that by M. Brault, of Algiers, I find no less than ten pages devoted to this complication, and numerous outbreaks in which the complication was noted are referred to from the days of Sydenham to recent years; most of these, however, were met with in temperate climates. This is in marked contrast with the silence on this subject in even the most recent articles on dysentery in English text-books, *e.g.*, neither in the latest edition of Manson's "Tropical Diseases," nor in the article on dysentery by Mr. Cantlie, in the first volume of Allchin's "Manual of Medicine," is any such complication even hinted at. In the many hundred cases of dysentery which I have treated I never remember to have met with it except in the one mentioned above.

It is not likely that such a phenomenon as described by the French writers could have been overlooked by English observers, so I suppose we must conclude that it is a complication of the dysentery of temperate climes rather than of tropical dysentery. M. Brault, however, ranks it with liver abscess as one of the "most classical and most habitual complications of dysentery."

Yours, &c.,
W. J. BUCHANAN, M.B.,
Major, I.M.S.

Bhagalpur, Bengal,
October, 1900.

THE SPECIFIC ORGANISM OF BLACKWATER FEVER.—In the *Deutsche Med. Woch.*, October 4, 1900, Dr. Hans Ziemann states that in his researches he has failed to find a specific organism in blackwater fever cases. In this he is at variance with Yersin. Dr. Ziemann finds that malarial attacks predispose to attacks of blackwater fever in certain districts. The æstivo-autumnal parasite, or what is termed the small parasite, met with in tropical malaria, seems especially to predispose to blackwater fever attacks.

British Medical Association.

A DISCUSSION ON ANKYLOSTOMIASIS.

(*Continued from p. 77.*)

III.—Leonard Rogers, M.D., M.R.C.P., I.M.S., Officiating Professor of Pathology, Medical College, Calcutta.

The very great differences of opinion on the subject of ankylostomiasis which the recent literature of the subject reveals affords ample reason for this debate. For example, while Mr. Thornhill[1] has frequently written most eloquently on the terrible ravages caused by this parasite in Ceylon, that careful observer, the late Dr. Macdonald,[1] wrote of the same colony that in a great number of cases the worm does very little harm, although capable under certain conditions of destroying life. Unfortunately Dr. Thornhill gives very few figures in his extensive writings, and on turning to the Ceylon medical reports for from 1886 to 1893, I was astonished to find very little reference to the disease in them; while, as I have elsewhere shown,[2] the figures given indicate a great decrease of the disease during recent years in that colony; while in Thornhill's own district, among a population of 160,000 persons, the recorded death-rate from this disease in the hospitals was but thirty-two in 1894; so that the evidence of these reports strongly supports the moderate views of Macdonald rather than the extreme ones of Thornhill. The same thing is seen in Assam, where ankylostomiasis was first discovered by Dr. Ruddock, who is of the opinion that a healthy person can support a fairly numerous colony of them (ankylostoma) without damage, and the parasite only assumes importance when the patient is lowered by some other disease;[3] while Dobson has recorded instances of perfectly healthy men harbouring from one to two hundred of the worms, and finding small numbers in over 60 per cent. of healthy Assamese, and in upwards of 80 per cent. of healthy natives from Bengal and other parts of India, asks, "At what stage does the parasite become injurious?" On the other hand, Major Giles wrote with regard to the terrible disease kala-azar, which has certainly carried off at least one-fifth of the population of a tract of country 200 miles in length, "All I wish to convey is that the increased mortality is due to ankylostomiasis and to no other cause."[7] And again, "What, then, is kala-azar? Kala-azar is ankylostomiasis," with no qualification whatever. In another passage he wrote that the disease was ankylostomiasis, acting in a population which "has for generations been continuously poisoned by malaria;" but fortunately it is unnecessary to discuss this question now that so high an authority on malaria as Major Ross,[4] after a short personal investigation of kala-azar in Assam, has recorded his opinion that "I think, then, with Rogers, that kala-azar is malarial fever," and further "that in the latter epidemics studied by Rogers this fever is also communicable from the sick to the healthy;" and again, "I agree with him (Rogers) that kala-azar is not ankylostomiasis"—although he adds that in several instances these words amounted to a serious complication, as I had previously stated was sometimes the case. Moreover, since Ross' confirmation of my conclusions on the subject, Major Giles has admitted[9] that—"and I am thus reduced to the conclusion that the disease now called kala-azar is something quite different from what was shown me," which should be read in connection with the following statement in my original report: "Dr. Giles's opinion that fever was not a common or a marked feature of the disease is accounted for by the fact that he was unfortunate enough to happen to commence his clinical work in Gauhati late in November, which is just the very time when the fever is at a minimum, and remains so until the commencement of the next rains, by which time Dr. Giles was engaged on microscopical work in Shillong" (a hill station which kala-azar never reached); "so that he only studied the disease at the season when most of the cases which had survived the previous rains have lost their fever, and fresh infections are at a minimnm." As far as I know, Mr. Thornhill remains the only advocate of the view that ankylostomiasis is the essential factor in the cause of kala-azar; and as he admits he has never seen a case of the disease, and his extreme views on the ravages of the disease in Ceylon are not accepted by his colleagues there, his opinion cannot be allowed any weight against the accumulated positive evidence, and now practically unanimous opinion, of all who have seen the disease in Assam, that it is purely malarial; while the successful eradication of the disease from tea gardens by the methods advocated in my report, which has been recorded elsewhere, may also be fairly claimed as to the soundness of my conclusions."[11 12]

The Sources of Fallacy.

How can the above differences of opinion be explained? This question has been answered by the researches of Dobson,[1] for Giles furnished no evidence in his report that he ever controlled his observations by examining healthy persons for the ankylostoma, and appears to have been quite ignorant of the nearly universal prevalence of the worm in them (although previous to his investigation Dr. McConel of Calcutta had found the worms in the great majority of *post mortems* in Calcutta as far back as 1882), and he gives no evidence to show that these parasites were met with in larger numbers in kala-azar cases than in healthy persons in Assam (which I subsequently showed they were not); yet soon after Giles's report was published, Major Dobson, as a result of examining over 1,000 persons for the worms by the thymol method, showed that they were present in some 67 per cent. of healthy Assamese, and 84 per cent. of several hundred healthy coolies who had been passed by medical officers as fit for emigration from Bengal, Madras, Chotta Nagpur, the Central Provinces, and the North-West Provinces; in short, a large part of India. I also found them in 66 per cent. of healthy Assamese, in slightly larger numbers than in kala-azar cases, and showed that in the latter disease less than twenty of the worms were present in upwards of 80 per cent. of the patients, while Thornhill[1] admits that "50 of these worms is a number altogether too small to have any deleterious effect," and the same writer agrees with others in considering that in order to produce anæmia at least 500 of the worms must be present for from six months to a year; with which I also agree. Another writer[5] has recorded "Short notes of 130 cases of ankylostomiasis," in which it appears probable that only eight cases passed more than 50 worms after thymol, while certainly three-quarters of them had less than that number present. The cases were admitted to hospital for all causes, from measles to locomotor ataxy, while 31 of them were admitted for malaria, and 19 more had malarial symptoms. Yet because they passed a few worms after thymol they are called cases of ankylostomiasis! At this rate upwards of 80 per cent. of the healthy inhabitants of large provinces of India suffer from ankylostomiasis! I have no hesitation in saying that nearly all the confusion with regard to this disease has arisen owing to the loose way in which the term ankylostomiasis has been frequently used.

Definition and Classification of Ankylostomiasis.

In order to avoid this source of confusion, I shall in this paper use the term ankylostomiasis to indicate a disease characterised by anæmia produced by long-continued small losses of blood through the gastro-intestinal mucous membrane caused by the presence of several hundreds of ankylostoma acting for many months; or by a still larger number acting for a shorter time. The very fact that in the St. Gothard tunnel outbreak as many as 2,000 to 3,000 of those worms were found in some cases which had suffered from anæmia for several months is alone enough to show that very small numbers of the worms cannot produce any appreciable effect on the system. That the abuse of the

term ankylostomiasis is a matter of practical importance is shown by an instance, which I have elsewhere recorded,[6] in which out of 72 consecutive cases which were treated by thymol for anæmia in an Assam dispensary, 17 died, no fewer than 8 of which succumbed within six days of the last dose of thymol, although the average number of worms passed by them was but 5 and the maximum 10 in these 8 cases, while not one out of the 72 cases passed over 50. The fatal cases, as well as the great majqrity of the others, were suffering from malarial cachexia, while many had diarrhœa or dysentery. Yet because they were anæmic, all were given drastic doses of thymol, and the end of some at least who might otherwise have recovered was certainly hastened by the treatment.

Most writers on this subject admit that, in order to produce anæmia, at least 500 ankylostoma must be present for from six months to a year, although several thousands would doubtless produce symptoms in a shorter time, but the latter are rare. It is to these classes of cases that I would limit the term ankylostomiasis, and using it as defined above I may say that the disease is rare in Assam villages apart from tea gardens or railway coolies, in the Nowgong district at any rate. I have met with several cases which I at first sight took the anæmia to be of this class, but which close investigation showed other causes, such as Bright's disease, bad feeding, malarial fever, syphilis, etc., to have been responsible for the condition.

On the other hand, there is another class of cases in which anæmia due to some other cause is complicated by the presence of from one to three hundred ankylostoma, the drain caused by which alone might be withstood by a healthy person under favourable circumstances for a long time, instances of which I have recorded in a previous paper, but which when added to other debilitating and anæmia-producing causes becomes an important pathological factor. These constitute the greater number of cases in which the parasite is seriously injurious in my experience, but they should be classed under the heading of the primary disease, malaria, dysentery, syphilis, &c., with the qualification that they are complicated by a harmful number of ankylostoma. Lastly, healthy men or those suffering from common diseases should not be classed as ankylostomiasis just because they happen to harbour a few of these parasites, the very small loss of blood caused by which, amounting to but a few drops daily, is repaired by the system without any ill effect whatever, for I have shown elsewhere that there is no reduction in the hæmoglobin or red corpuscles in such cases.

The Differential Diagnosis of Ankylostomiasis.

In advocating above the careful restriction of the term ankylostomiasis to its proper meaning of disease produced by this parasite, it is very far from my wish to minimise the importance of the *rôle* played by the worm in various parts of the world, of which I am fully convinced, but rather desire to lessen the confusion which hampers efficient action to lessen the scourge, for such a disastrous result as that referred to above can only bring discredit on the thymol treatment, and so lead to its being omitted in suitable cases. What is wanted is more careful diagnosis, a matter of no small difficulty, as experience has taught me, for although the number of ova in the fæces is a sure guide to the presence of large numbers of these parasites, the absence of them does not prove that the anæmia has not been caused by previously present worms, for they may have dropped off, but in that case thymol will be unnecessary. Moreover, I have found that ova can be easily found in a single cover glass preparation of the fæces when but five to fifteen worms are present; but, on the other hand, if they are met with in nearly every field of the microscope, then it is certain that the parasites are present in active numbers, and thymol is indicated if the patient is in a condition to stand it. But the mere discovery of one or two ova is not an indication for thymol unless it is quite certain that the drug will do no harm, or much greater injury may be inflicted than will be compensated for by the removal of the very few worms which may be present. This remark applies especially to cases of chronic malaria, in which I have recently found a condition of pigmented atrophy of the mucous membrane of the small intestine, which I am convinced is a very important factor in fatal cases with intractable diarrhœa, and which very probably accounts for the low fever seen in these cases, which may be caused by the absorption through the thinned intestinal mucous membrane of more poisonous material than the liver can adequately deal with. It is in these cases that thymol is so dangerous.

The clinical features of ankylostomiasis are too well known by workers in tropical diseases to need description, but they may be very closely simulated by Bright's disease, the œdema in which is so similar, while albuminuria not unfrequently appears in the most advanced stages of ankylostomiasis. Very similar, too, is the anæmia produced by bad feeding, more especially a deficiency of nitrogenous food, an example of which I have elsewhere recorded.[3] Cases of both these diseases I have myself mistaken for ankylostomiasis until I made the blood examination to be mentioned immediately. In the anæmia of dysentery the patient is usually very wasted as opposed to the fat appearance in the worm disease. Most important of all is the differentiation of malarial anæmia from that due to ankylostomiasis, and although in typical cases the clinical pictures are in marked contrast (a table of the differences between which I have published in a previous paper[2]), yet in certain cases, more especially when the two diseases complicate each other in various degrees, it is well nigh impossible to solve the problem of their relative importance by clinical means alone. Yet it is just in these very cases that it is most important to do so, for not only is thymol particularly dangerous in advanced malarial cachexia, but the treatment of the anæmia of the two conditions is quite distinct. Fortunately a way of differentiating them has been discovered in the essentially different type of anæmia in the two conditions, which I found to be of the greatest value in the solving of the difficult problems surrounding the mysterious kala azar, which largely resolved itself into deciding whether malaria, or ankylostomiasis, or both together, were the cause of the anæmia which all workers acknowledged to be present in the disease.

The Types of Anæmia in Ankylostomiasis and Malaria.

In such a country as Assam, in which the majority of healthy persons harbour a certain number of ankylostoma, while in the latter stages of ankylostomiasis few or none of these parasites may be present, it is exceedingly difficult, if not altogether impossible, to decide from the number of worms found in a given case what part they have played in the production of the anæmia, while I have just shown that clinical evidence may also be at fault in these cases. The problem, then, was a very difficult one, but it appeared to me that the type of the anæmia produced by chronic malaria, in which the red and the white corpuscles of the blood are destroyed, but the heamoglobin is largely retained in the body in the form of pigment in the liver, spleen, &c., might be expected to differ from that produced by the slow hæmorrhage which is the essential feature of ankylostomiasis. The examination of such literature as I could obtain in the Calcutta Medical College Library, however, revealed such a hopeless difference of opinion as to the type of the anæmia of more especially ankylostomiasis, that I determined to make as full an examination of the blood as possible in both diseases. The results exceeded my most sanguine expectations, for not only did the type of the anæmia in the two diseases prove to be essentially different but the differences were found to be so constant as to furnish a certain means of diagnosis between the two diseases, and even served to indicate the presence of an injurious number of ankylostoma in a case of typical malarial

fever when the anæmia was so slight as to be only detected by a quantitative examination of the blood.

The details of this research have already been published, but their practical importance is so great that I gladly avail myself of this opportunity of bringing them before a larger audience, so will briefly describe them. In the first place, it must be mentioned that I found the blood of healthy inhabitants of Assam to be very different from the European standard. Thus, although the numbers of the red and white corpuscles were about the normal, the percentage of hæmoglobin averaged only 62 per cent. of Gowers' standard, in spite of all cases in which ankylostoma might have been a disturbing factor having been carefully excluded. The colour index in these healthy cases (men) worked out at 0·65, which must be borne in mind in studying the figures in the different forms of anæmia, which are given in the accompanying table:—

—	Percentage of Hæmoglobin	Red Corpuscles per Cubic Millimetre	White corpuscles per Cubic Millimetre	Ratio of White to Red	Specific Gravity	Hæmoglobin Value or Colour Index
Healthy Assamese	62	4,734,000	7,325	1:684	1054	0·65
Epidemic malaria of Assam (kala azar	33·45	2,462,000	2,600	1:1170	1048	0·65
Ordinary chronic malaria	31·6	2,000,000	1,600	1:1400	1042	0·73
Ankylostomiasis	15·2	1,145,000	5,338	1:524	1034	0·31
Malaria + ankylostomiasis	37·4	3,120,000	3,200	1:975	1039	0·43

Secondly, the average blood estimation in ordinary cases of malarial cachexia in a part of Assam which was unaffected by the epidemic, malarial fever, or kala-azar, which are given in the third line of the table, show a nearly equal reduction of the red corpuscles and the hæmoglobin, so that the colour index is 0·73, or slightly higher than in healthy Assamese. Further, the white corpuscles are greatly reduced, both absolutely and relatively, to the red, so that there is only 1 white to 1,400 red, or about half the number in healthy people. The specific gravity is also reduced, but only to a slight degree in proportion to the anæmia. Precisely similar conditions were met with in a large number of cases of the epidemic malaria fever of Assam, as is shown in the second line of the table, which fact alone, contrasting as it does with the condition met with in ankylostomiasis, is sufficient to indicate that kala-azar is essentially malarial. In these cases the colour index is precisely the same as in the healthy people of the province, showing that the hæmoglobin and the red corpuscles are equally reduced, and the colour index is 1, allowing for the low amount of hæmoglobin met with in these people. Again, the white corpuscles are reduced much more than the red, so that there the proportion is 1 to 1,170. The specific gravity is but slightly reduced in proportion to the anæmia. The figures given by Cabot and by Waddell[15] in India in cases of chronic malarial anæmia agree very closely with those recorded above, so that it is evident that the anæmia of the Assam epidemic is identical with that of malaria in other places.

We may now turn to the conditions met with in ankylostomiasis, with regard to which there is so much difference of opinion and positive error in even the most recent works on tropical diseases. A glance at the fourth line of the table shows at once the contrast between the tpye of the anæmia in the worm disease and that of malarial cachexia. The most important distinction is that in ankylostomiasis the hæmoglobin is reduced twice as much as the number of the red corpuscles, so that the colour index sinks to 0·31, or about one-half of the normal in healthy people of Assam. Again, the white corpuscles, although absolutely reduced in numbers, are relatively less reduced than are the red, so that the proportion rises to 1 to 524 against 1 to 684 in the healthy controls; a change which I subsequently found had also been noted by Lutz. Once more the specific gravity of the blood is greatly reduced, having averaged only 1,034, the reduction being greater in proportion to the degree of the anæmia, as estimated by the percentage of hæmoglobin, than it was in malarial cases of an equal degree. Thus in every point the type of the anæmia of the two diseases is radically different. But this is not all, for it is not sufficient from the diagnostic point of view to show that the average changes differ unless it can also be proved that the extremes met with in either condition do not overlap to any extent those met with in the other. As a matter of fact, I have found that they do not overlap at all in the case of the essential point, namely, the colour index, for the lowest point met with in uncomplicated cases of malarial anæmia was always over 0.5, while the highest encountered in ankylostomiasis was always under 0.4 in Assam, so that this point serves as a certain means of differential diagnosis between the two forms of anæmia. In view of the statement in recent textbooks on tropical diseases, that the hæmoglobin value of the corpuscles is not depressed correspondingly to the fall in numbers—a statement which appears to have been copied from one book to another—I may be asked if there is any confirmation to form the results recorded above. There is, for Dr. Sandwith of Cairo, in a valuable paper on ankylostomiasis in Egypt, gives estimations of the hæmoglobin and the number of the red corpuscles in 178 cases of the disease, which show the percentage of hæmoglobin to be reduced nearly twice as much as that of the red corpuscles, so that the colour index was reduced to about one-half, just as it was in my own cases, allowing for the abnormally low hæmoglobin value of healthy Assamese. Further, I have received communications from several workers in India to the effect that they had been able to confirm my observations on these points.

Lastly, in a few instances of malarial cachexia (under 7 per cent. of kala-azar cases) there were sufficient of the worms present to constitute a definite factor in the production of the anæmia, and in all of them the blood changes were found to be intermediate between those of the two primary conditions, as is shown in line 5 of the table. One of these cases is of particular interest. It was an early case of the epidemic malarial fever in which no clinically evident anæmia had yet appeared, but in which the spleen and liver were enlarged. On examining the blood the hæmoglobin value or colour index was lower than usual in pure malarial anæmia, so I at once told the medical man in charge of the case that I suspected the patient to harbour an active number of ankylostoma, and proposed to give him thymol. This was done, and 159 of the worms were passed. In this case, then, the blood test was found to be delicate enough to detect the presence of a not very large number of the parasites before even clinically evident anæmia was present. Further, I have found the characteristic alterations to persist for many months after the removal of the worms, especially in the absence of proper treatment by complete rest and iron, and in one case in which a patient has been successfully treated for ankylostomiasis, but was subsequently attacked by kala-azar, a colour index intermediate between those typical of either disease was found although no worms were present, showing that the effect of the previous attack of ankylostomiasis on the blood had not completely passed away. Now the duration average of kala-azar cases is but seven months (against several years in ankylostomiasis as a rule), so it is evident that in any case of the epidemic fever in which ankylostoma have played a definite part in the production of the anæmia, their action will be at once revealed by the approximation of the type of the anæmia to that of ankylostomiasis, as evidenced by a low colour index, even if all the worms have already dropped off. Yet in less than 7 per cent. of the cases was this found to be the case, which alone is complete proof that ankylostomiasis is not an

essential or even a common factor in the production of the anæmia, which is found by all observers to be a constant feature of kala-azar, and hence the worms cannot be a primary cause of the disease itself. Other examples of the blood examination in differentiating forms of anæmia are given in my report.[3] One was a man who had a large liver and spleen, but who said he had not suffered from fever recently, while in some respects he resembled a case of ankylostomiasis, so that it was impossible by clinical means to decide by which disease his anæmia had been caused; the blood estimations showed the ankylostomiasis type, which was therefore diagnosed and subsequently was found to be correct and successfully treated.

It only remains to briefly explain the differences of type in the anæmia of the two diseases, and to point out the therapeutical indications to be derived from them. Firstly, with regard to the colour index, it will be at once evident that the differences noted are just such as might be expected, for in the case of ankylostomiasis the hæmoglobin is being continually slowly drained out of the system, and while it can only be replaced with great difficuly, especially in the case of people who are living on purely vegetable diet, while on the other hand the red corpuscles are being rapidly formed in the bone marrow, for I have found the yellow marrow of the shafts of the long bones to be converted into red marrow both in ankylostomiasis and in malarial anæmia, just as it is in pernicious anæmia. The red corpuscles being more easily and rapidly replaced than the hæmoglobin, the colour index or amount of this substance in each corpuscle must necessarily fail, just as it does in all forms of slow hæmorrhage. In malaria, on the other hand, the hæmoglobin is not lost to the system, but is stored up in an organic combination in the liver and spleen, &c., from whence it can be absorbed to restock the red corpuscles as they are turned out. This process probably accounts for the occasional disappearance of the black pigment from these organs in the later stages of the more chronic cases of kala-azar, which has been pointed out by Ross,[4] especially those who are tending toward recovery. The decrease of the white corpuscles in the malarial anæmia is doubtless due to the constant fight that they are carrying on with the parasites in the blood, as indicated by the slight leucocytosis which may occur, while in ankylostomiasis, on the other hand, they are not especially destroyed, while they are more easily replaced than are the red corpuscles, and hence a relative increase of the white cells occurs. It should be mentioned that in malaria the white corpuscles should be counted during the absence of fever, or else they may appear to be normal when greatly deficient, owing to the occurrence of a relative temporary leucocytosis during fever. The lower specific gravity of the blood, relatively to the degree of anæmia in ankylostomiasis, than in malarial anæmia is explainable on the ground that in the former disease there is a steady loss of albuminous constituents of the blood, so that the blood becomes more watery. In this connection it is worthy of notice that I found hæmic murmurs to be much more common in the anæmia of ankylostomiasis than in that due to malaria of similar degrees—a fact which supports the theory which attributes these murmurs to alterations in the specific gravity of the blood. All the changes met with, then, are just those which might have been expected to result in either case.

Finally, the correct treatment of these two diametrically opposite forms of anæmia should obviously be different, for in that due to ankylostomiasis iron is urgently indicated in order to assist in the production of hæmoglobin, while arsenic will be of less importance, as it is not much use stimulating the output of red corpuscles when there is no hæmoglobin with which to stock them. In the anæmia of malaria precisely the opposite holds, for there is no urgent necessity to pour iron into the stomach, very little of which will be absorbed (Burge), when there is already a large stock in an organic combination ready to hand in the liver and spleen, while on the other hand arsenic and bone-marrow tabloids (the latter of which more especially I have found to be of extreme value in malarial cachexia) are strongly indicated in order to increase the output of the red corpuscles which are being or have been destroyed by the fever. These indications I have followed with great success in many cases, and although they are often carried out as a result of empirical knowledge, a rational basis is afforded them by the research which I have described.

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[1] "First Indian Medical Congress Report."
[2] "The Epidemic Malarial Fever of Assam or Kala-azar," a reply to criticisms, Rogers.
[3] "Report on Kala-azar," Rogers.
[4] "Report on Kala-azar," by Ross.
[5] "Short Notes on Ankylostomiasis," by Otho Galgey, of St. Lucien.
[6] "The Distribution and Harmfulness of the Ankylostoma," Rogers, JOURNAL OF TROPICAL MEDICINE, October, 1898.
[7] "Report on Beri-beri, or Anæmia of Assam," by Major G. M. Giles.
[8] *Indian Medical Gazette*, January, 1898.
[9] *Indian Medical Gazette*, 1899, p. 91.
[10] "On the Epidemic Malarial Fever of Assam, or Kala-azar," *Med.-Chir. Trans.*, vol. lxxxi. 11.
[11] *Ibid.*, vol. lxxxii.
[12] *British Medical Journal*, September 24th, 1898.
[13] *Jour. of Path. and Bact.*, December, 1898.
[14] *Indian Medical Gazette*, 1883.
[15] Translation of Lutz's paper on "Ankylostomiasis" in Kynsey's "Beri-beri of Ceylon."

(To be continued.)

New Drugs, &c.

we have prescribed them with benefit. The Maltine Company have combined Maltine with several ingredients in addition to cod-liver oil—with hypophosphites, with yerbine (the active principle of yerba santa), and with phosphates. We believe that Maltine is employed in some form by every practitioner in the tropics, thus falsifying the belief that such medicines are unnecessary in the tropics, and it is our opinion that it should be more widely used during the cold seasons than it is at present.

News and Notes.

Blood Letting in Heat Stroke.—A case of heat stroke under the care of Dr. C. Klein, and related in the *Münch. Med. Woch.*, July 3, 1900, is interesting on account of the immediate benefit which ensued from the abstraction of 8 ounces of blood by venesection. Many other remedies had been tried, but venesection proved immediately and permanently beneficial.

Return of Drs. Low and Sambon.—A hearty welcome has been accorded to Drs. Sambon and Low on their return from their sojourn in the Roman Campagna. Their official report is not yet to hand, but the general result of their stay is well known. Neither of the two *devotés* to science have contracted malarial fever, and we are glad to note that both gentlemen seem in excellent health.

We are pleased to learn that Mr. G. B. Warren, Laboratory Assistant, London School of Tropical Medicine, has quite recovered from the fever contracted from the bites of the malarial infected mosquitoes sent to the school from the Roman Campagna.

The Germ of Yellow Fever.—From researches being conducted in Cuba considerable doubt is being thrown upon the potency of the so-called Bacillus icteroides (Sanarelli) in the etiology of yellow fever. Agramonte states that the bacillus in question has no more to do with the production of yellow fever than has the common Colon bacilli, and the same observer states that he has succeeded in isolating Sanarelli's bacillus in bodies in which death was not due to yellow fever. Lutz is of opinion that a mistake has been made in attributing specific pathological influence to Sanarelli's bacillus.

A Specific Organism in Dysentery.—In the *Deutsche Med. Woch.*, October 4, 1900, Dr. W. Kruse reports upon investigations on the cause of dysentery. During the prevalence of an epidemic at Laar Dr. Kruse met with an organism allied to the typhoid and Colon bacilli. It is shorter and thicker than the typhoid bacillus, and is not flagellated. Unlike the Colon bacilli, the organism isolated by Kruse in dysentery failed to produce fermentation in a grape-sugar medium.

Letters, Communications, &c., have been received from:—

B.—Dr. R. C. Bennett, Trinidad. Dr. W. L., Braddon, Negri Sembilan. Major W. J. Buchanan, I.M.S., Bhagalpur.

C.—Lt.-Col. O. H. Channer, Belgaum.

E.—Miss Edith Wynne Edwards, L.R.C.P., Cawnpore. Dr. P. G. Edgar, Perák.

H.—Dr. A. H. Hanley, Bonny.

M.—Dr. Preston Maxwell, S. China. Dr. Neil Macleod, Shanghai.

N.—Col. J. H. Newman, I.M.S., Gowran.

S.—Dr. Henry Strachan, West Coast. Dr. R. A. Shekleton, Bonny.

T.—Dr. Chas. Todd, Mashonaland

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Hongkong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
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1.—All communications will be acknowledged in the Journal under the heading "Letters and Communications Received." Contributors who do not see their names in the list should communicate forthwith with the Editors or Secretary.

2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the Journal of Tropical Medicine should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."

The Journal of Tropical Medicine.

CONTENTS.—DECEMBER, 1900.

Original Communications.

PANI GHAO — WATER-SORE — COMMONLY CALLED "SORE FEET" OF ASSAM COOLIES.

This disease I met with and studied during my three years' service as Medical Officer to the Assam Frontier Tea Company of the Dibrugarh district of Assam.

I had five gardens, with nearly eight thousand working coolies under my charge, five hospitals aggregating 230 beds, with dispensaries, native doctors, &c., so that I had ample opportunities of studying the disease in all its stages.

It is not a serious disease from a vital point of view, as it is never fatal, but it causes a great amount of pain and discomfort to the patient, and is a matter of much importance to the planter, as it incapacitates so many coolies from doing their work. During its season—from May to October—more coolies are laid up from this cause alone than from any other, with the exception perhaps of malaria.

The cases may amount to as much as 5 per cent. of the total working force after heavy rain, dwindling to 1 per cent. in a dry spell. These dry and rainy spells alternate throughout the tea Season, the climate of Assam differing in this respect from that of India proper, where the "rains" and the "hot weather" are two distinct seasons.

The treatment constitutes a large part of the surgical work of the Tea Garden Hospital, and demands much care and attention, not only from the doctor on his weekly or bi-weekly visit, but from the manager on his daily round. It is common enough in Assam, but I cannot find that it has been described before. It is universally known amongst Europeans as "sore feet," sometimes as "water-sore." The Assamese call it Boree Ghao—foot sore—and the coolies a phonetic corruption of that word, or else they translate it each man into his own language. These latter are many, and the coolies are indeed foreigners in the land and strangers to one another.

The illustrations have been drawn by me from original sketches taken from nature, and the short clinical notes on them were in nearly every case taken at the same time.

Symptoms.—These are simple enough, and the condition is easily recognised. The first symptom is one of intense itching with a little pain, which increases on the second day, when an eruption begins to appear and the foot swells. Walking now becomes painful. The eruption is of a distinctive character, and by its position and appearance we are able to divide the disease into three heads for convenience of description:

(1) The Vesicular or Pustular Form.
(2) The Herpetiform Variety.
(3) The Interdigital Variety.

1.—The Vesicular.

This, the most common form, is found both on the plantar and dorsal aspects of the foot, but, except in rare cases, it is not found above the ankle, a statement which applies equally to the other forms. The eruption first appears as small dusky red spots under the skin fig 1), which next day forms vesicles, measuring from two to five millimetres in diameter, and numbering from three or four to a dozen. They are placed at irregular intervals, generally from a quarter to three quarters of an inch apart, and, as a rule, we find them in colonies. One group may occupy the line of junction of the plantar and dorsal surfaces on the

inside of the foot, from the heel to the ball of the big toe; in another case the whole of the dorsum will be found studded with vesicles (fig. 2), or the whole of the plantar surface. Sometimes the whole foot is found covered with vesicles, in some cases all of one age, sometimes partially healed ulcers, or pustular blebs, are found interspersed with new vesicles (fig. 3).

2.—Herpetiform Variety.

Here the vesicles are nearly always situated on an inflamed base, and instead of being isolated are close together, and have a tendency to run into one another, and they may present the appearance of a number of vesicles dotted close together (fig. 4), but in most cases these are large and branching, resembling nothing so much as the crests of a mountain range as depicted in an ordinary map (fig. 5). We generally find a central mass of vesicles on a swollen base, with several outlying vesicles (fig. 6), or we may get two centres of disturbance (fig. 7). I have never seen the preliminary dusky red spots in this variety, the intense itching and pain of the first day being followed, on the second, by full blown vesicles on an inflamed base.

Fig. 1.—Vesicular Form.

Small dusky red spots, at junction of dorsal and plantar aspects, at inside of left foot. 1st or 2nd day.

This œdema is, I believe, in this instance, primary, not secondary; it is different to the general swollen condition of the foot caused by undue exercise, as it occurs at the beginning and is confined to the immediate area of the vesicles.

The peculiar eruption is formed by the fusion of many vesicles, but why this distinctive formation is assumed is not easily to be understood. There are no septa, and if the vesicles are opened the fluid drains away easily.

It is only found on the dorsum or the dorso-plantar junction, and is most often seen on the anterior portion of the dorsum. The prodromal itching and pain, œdema, vesicular eruption and subsequent ulceration constitute an outward resemblance to a case of herpes zoster, allowing, of course, for difference of position and altered conditions; and it is for this reason that I have called the variety herpetiform, though I have no reason to suspect a nervous origin.

3.—Interdigital.

This is probably a division of the vesicular variety, the vesicles being discrete, not confluent, but the position of the interdigital areas, with their loose skin, moist surfaces, and tendency to catch and retain foreign matter somewhat alters the course of the disease and renders ulcers and abscesses more common and more difficult to heal. Abscesses resulting from "sore feet" are nearly always found in this region. Another reason for putting it under a different heading is that the cultivators of Bengal suffer from a vesicular eruption of a similar character, but confined to the toes, the herpetiform variety being unknown. The Bengal variety is mild in type and consists of tiny watery blebs under and between the toes, such as I have often seen in Assam.

One day in hospital I had just opened a nasty

Fig. 2.—Vesicular Variety.

Yesterday, foot began to burn, small whitish spots now seen, some have had the skin removed from top of vesicle and show slightly red. 2nd day.

abscess between the toes (fig. 8), and a Bengali clerk, who happened to be there, told me he had seen a similar case in his country, but I believe it to be very rare there. Their remedy is to wash the foot, oil it, and then hold it in the smoke of a wood fire. The course of the disease after the appearance of the eruption depends on the way it is treated. If the parts are rested and kept surgically clean the sores will soon heal, but if the coolie goes on working instead of coming to hospital, or walks about in his lines, getting firewood, tending cattle, &c., &c., his feet get no rest, the parts are not kept clean, and the results are more serious—ulcers are formed, abscesses appear, and the whole process of healing is retarded. These ulcers, unfortunately, owing to the restless

habits of the coolie, have a tendency to become chronic. It may be necessary to state that a coolie never wears boots or shoes of any kind.

Pathology.—Here and there in the part affected isolated papillæ become enlarged, infiltrated and painful. On the second day serum exudes from the papillæ and separates the epidermis from the true skin, forming a vesicle, which in the thin-skinned regions of the sides and upper parts of the foot is easily recognised, but is hardly noticeable beneath the hard thickened skin of the sole. On the third day the clear fluid begins to be infiltrated with leucocytes and a small pustule is formed, as large as a small split pea, which, being opened, the skin will be found to be separated beyond the apparent limit of the pustule. On the sole of the foot the pus will sometimes spread out in a layer between the dermis and epidermis, forming an irregular bleb half-an-inch or more in diameter, generally more or less rounded, but sometimes elongated with the long axis across the foot (fig. 9). If the vesicle is opened and kept surgically clean before the contents have been purulent it will simply dry up and leave no scar, the true skin being hardly affected. But if it is not treated, or the part is not rested, or if foreign matter gets in after it has been opened, the skin and subjacent tissues become infiltrated with leucocytes, the tissues break down, pus is formed, and the result is a deep-seated abscess, or an ulcer of varying size and intensity (fig. 10). These abscesses are most commonly met with in the interdigital variety. It will often be seen, that though a purulent bleb is in the main superficial, the mischief has bitten more deeply in places (fig. 11). The ulcers result in cicatrices varying in size and thickness, but rarely larger than a shilling. In some cases they are at first raised, as in cases where the patient does not take due care of himself, and walks about too much—the tissues round the ulcers become infiltrated and organised, a sort of fibrous ring is formed, and they heal slowly (fig. 12). In a few cases the ulcers take on a virulent form and spread both laterally and deeply, with much destruction of tissue, but I believe this to be accidental, and to be similar in origin to the terrible ulcers known in Assam as the Naga sores. (These latter are akin to Bagdad boils or Aleppo sores, only they attack the feet and legs. I have seen them extending over the whole dorsum of the foot and two or three inches up the leg, the tissues being eaten away down to the ligaments of the joints.) There is often infiltration of the tissues surrounding a group of vesicles, or the resultant ulcers; leading to partial arrest of the circulation and œdema, either of the foot as a whole or of the region to which the vesicles are confined. I believe this is generally caused by want of rest, but in the herpetiform variety it is primary and limited in area.

Fig. 3.—Vesicular Type.

Whitish rounded vesicles are seen. On opening these, the contents will be found purulent, and the surrounding skin undermined. Two sores have been opened, and were of older date. 4th or 5th day.

Fig. 4.—Herpetiform Type.

Three days old, full, not much swollen, small vesicles, some run together, are seen instead of the usual branching type.

In the herpetiform variety, too, the vesicles run into one another at an early date, there being no septa, so that even on the second day a prick, or a couple of pricks, will cause the fluid to drain away from the whole of the vesicular patch; and the serum having been drained off and the epidermis removed, in favourable cases the result is little more serious than a large blister or burn of the first degree, though if there was much œdema, a little time must elapse before absorption takes place.

Ætiology.—This disease is, as far as I can ascertain, only to be found in the Province of Assam, and is more common in Upper Assam than in Sylhet and Cachar. It is found, not amongst the indigenous native population (not to any extent) but amongst the imported coolies who work on the tea gardens. The natives of Assam itself are not very fond of labour, and in any case are scattered and few in number, so foreign labour has been imported men and women with their families, varying in religion, caste and race, from the bazaar bred Aryan leather-sellers, to the sturdy aborigines of Chota Nagpur. These people when they reach Assam, are all called "coolies," and it is amongst these that I have studied pani ghao.

The Assamese suffer too, but not to any great extent; each man is his own master, his work is not continuous, the soil is bountiful, and so he is not exposed to the same conditions as the tea garden coolie who has to tend the tea bush all the year round. I have been told that they only suffer during the brief periods when the ground is being ploughed for the rice crops, and I also heard from some (Assamese villagers) that it only occurred when the men were cultivating soil quite near their villages, or round their own huts.

The cultivators of Bengal seem to suffer from the mild type of the interdigital variety, but not from any other.

I think that for the better understanding of the various theories concerning the cause and origin of Pani Ghao, a short description of the tea garden and the way it is worked is necessary, and the description will more particularly apply to the Talap and Dum

Duma districts of Lakimpur, where I worked in 1897, 1898 and 1899.

A tea garden is a clearance in heavy forest jungle, varying in size from five hundred to fifteen hundred acres, though of course where two or three gardens lie together, or where Assamese villages are adjacent, the clearances are very much larger. The country consists of flat alluvial soil, brought down by the Brahmaputra in former ages, and the tea bushes (which vary in height and diameter from three to five feet) are planted in rows, four feet apart each way, divided up into suitable blocks by roads and paths. The main object of tea planting is to secure as many tender leaf shoots from the bushes as possible. To this end the bushes are so pruned and plucked as to present a flat top for convenience of plucking; whilst in order to make them "bushy," and produce the

FIG. 5.—HERPETIC FORM. 19th, Began to itch. 20th, Began to swell. 21st, Eruption appeared. 5th day.

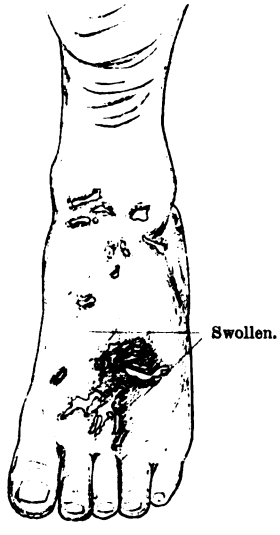


FIG. 6.—3rd day.

largest possible amount of "leaf," the soil between the bushes is kept in a constant state of cultivation, the garden being double hoed in the cold weather and single hoed once in two or three weeks during the tea season, a portion of the garden being hoed every day. By this operation the grass and weeds are hoed in and the soil kept open and loose, so that the rain may readily soak into it. The result is that the garden soil is always more or less like a ploughed field at home, and as the rain continues for days together, and sometimes for a fortnight without stopping, it is conceivable that the soil gets very sloppy.

The men as a rule do the hoeing and the women the plucking, though the former, when there is much "leaf," go out plucking when their hoeing is done, and the women hoe at the commencement of the season. The pluckers go out at 6 a.m., coming in at 11 a.m. to bring in their baskets and have their food, then out again from 12.30 to 5.30. The men go out earlier, and finish their hoeing task at from 8 to 10 a.m., according to physique; then they bathe and feed, and, if wanted, go out to pluck, weighing in their leaf in the afternoon. The coolies go barefooted both in and out of doors, and the result is that the skin of their feet becomes saturated and sodden by the wet earth, and the more rainy a season is the oftener this saturation takes place, and the more cases of "sore feet" we get. The women are more subject to this complaint because they are standing about for longer hours in muddy soil.

Pani ghao is more common in rainy weather, but whilst the heavy rain is actually coming down the soil is beaten flat, looking like the sands when the tide has gone out. The rainal so washes the coolies' feet, and it is when the spell of rain is passing off, and during the first few days of the hot spell which follows, that the new cases come in fastest, which seems to show that a certain consistency of the soil, not quite slushy, as well as a certain temperature, is conducive to "sore feet." Here we must note that cases do not come into hospital until the second day of the disease, as a rule, and we do not know what period elapses between exposure to infection (if I may use the term) and the appearance of the eruption.

The first cases of the season come in May, generally towards the latter end, and the last in December; but the worst months are June, July, August and September, and no cases occur in the cold weather, although we get some rain after Christmas.

I remember noticing that on one tea-garden, which from want of sufficient labour was not hoed so often, and therefore became overgrown with weeds and grass, the "sore feet" cases were few in number, but when the garden was brought into full cultivation again, and the people were standing all day in wet earth, cases became numerous. It was also noticed on the same garden that coolies working on soil recently reclaimed from the forest and planted with tea were more subject than coolies working in other parts of the garden. This may have been due to the light character of the soil, which becomes more easily slushy than the heavier soil found in the older part of the garden. This same soil, black and evil smelling, formed by the rotted vegetation of ages, fermenting in the close, moist jungle, is a veritable hot-bed of malaria, dangerous to coolie and European alike. Coolies working in it suffer heavily from malaria as well as from sore feet.

The coolies live in rows of huts arranged in groups called "lines," which may contain from 100 to 1000 or more inhabitants, and are usually bounded by a road or roads with a fence on the outside, the tea bushes coming up to the roads and close up to the huts.

Now, the sanitary customs of the coolie are oriental and primitive, and the soil for a considerable distance around the lines, especially if big ones or old ones, is always well manured. The lines themselves are not easy to keep clean, even with careful supervision. The coolie is naturally an unclean being, and objects to cleaning up; even on the same garden you will find one set of lines cleaner than another, perhaps because the lie of the land admits of easier drainage, or what is more likely, because in one case the soil is sandy and porous, and in the other heavier and more reten-

tive, so that insanitary matter of all kinds is retained on the surface instead of being carried down into the soil.

In seeking for the cause of pani ghao we must examine the conditions under which it may be found.

It occurs in the rainy season, more especially in June, July, August and September, which are the wettest and hottest months. It does not occur in the cold weather even when it rains.

It attacks the coolies only when they have been working in *wet* loose soil; grass-covered soil or smooth beaten roads do not cause it, neither does working in loose *dry* earth, as in the "cold weather" hoeing.

The number of cases increases after a heavy rain, and rapidly decreases during a hot spell.

Thus we see that two indispensable factors are heat and rain. That neither of these causes can act alone we see by the fact that cases do not occur when we have rain in the cold weather, or during the hot *dry* weather in April and May. That it is not due to a combination of heat and moisture (atmospheric) is shown by its absence amongst Europeans and amongst the tea-house coolies, who work under cover and are not concerned in the actual cultivation of the bush, and by the fact that various native races of Assam suffer only to a small extent, and then only when cultivating the soil under conditions somewhat similar to tea-garden culture.

Fig. 7.—Herpetiform Variety.

Two days ago there was slight swelling, slight heat, also pain and intense itching. 3rd day.

It is not due to some air-borne cause, for then one class (the cultivator) would not suffer and the others be exempt.

We find, then, that only the men, women and children actually working in the garden, walking slowly about with their naked feet on the bare, loose cultivated soil, are liable to be attacked. The thick wet mud sticks to the coolies' feet like a stocking, so that when they come in from work they look as if they had had their feet in poultices, the skin being white and sodden; and I believe this sodden condition is an indispensable preliminary. In this sodden condition the horny layer of the epidermis is swollen and altered in character, and would be easily permeated by anything in solution which might be applied to it.

It would seem that pani ghao is caused by some irritant of chemical, bacterial or mechanical origin applied to the foot during actual contact with the wet soil of the garden, or by the contact of the foot, whilst still sodden and receptive, with unclean matter in or around the lines. Three factors we know: (1) heat; (2) rain; (3) the wet soil of the garden; the fourth is yet to be found. The soil infection theory is the one usually held in Assam by planters and doctors,

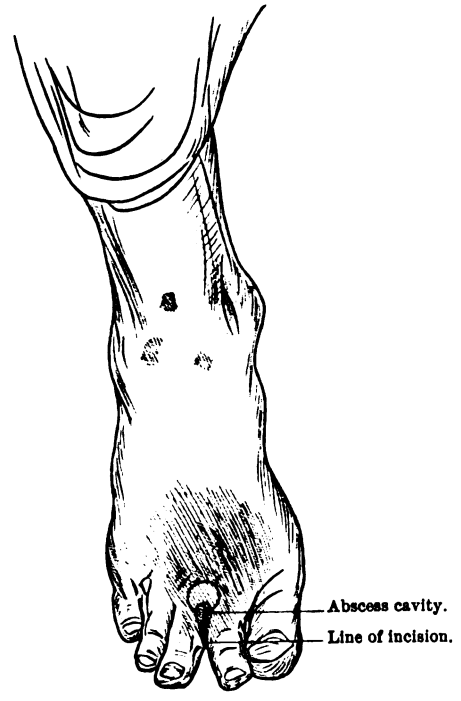


Fig. 8.—Interdigital Variety.

The abscess has been opened by an incision between the first and second toes. The skin peeled off the area and presented a light red colour.

but as far as I am aware the subject has not been gone into experimentally. The time and appliances at my disposal did not allow of any proper examination of the soil. The soil itself is sandy, with clay here and there; a belt of pure sand, forty to sixty feet thick, lies at a depth of from six to eighteen feet from the surface. The line infection theory is one to which I am drawn by one or two observations. The coolies themselves say that they suffer most when working in the garden immediately around the lines (where filth abounds). That the soil is actually affected by this may be seen from the fact that the tea bushes grow most luxuriantly in these situations, and along the main roads, where similar conditions prevail. It struck me that a coolie with sodden feet might pick up the disease in and around the lines and not in the garden at all. He must get his feet sodden in the

garden—it could hardly occur elsewhere—but when his work is done he is wandering about everywhere, and undoubtedly his feet come in contact with much unclean matter. The other coolies also wander about, but they are protected by the hard, horny impervious outer layer of the skin.

I collected some interesting statistics at Talap which seem to bear this out. The working coolies

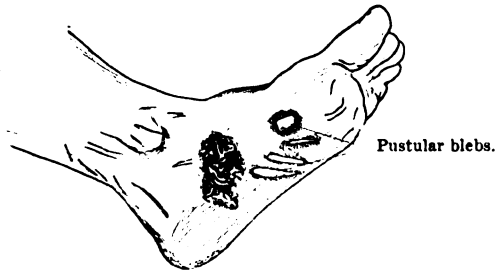


FIG. 9.—HERPETIC AND VESICULAR VARIETIES.

HERPETIC at dorso-plantar margin on inside of foot. 3rd day.

VESICULAR FORM.—Here seen as on sole of foot as transverse purulent blebs arising from vesicles of earlier date than those in herpetic patch. Probably 5th or 7th day.

number 3,000 (total population 5,000), and are housed in lines of various sizes, on different soils, some newly built, some old and scattered over a wide area, so that Talap may be taken as a fair sample of a tea garden

Name of Line	Description of Line	Per-centage
TALAP DIVISION:		
Old	Crowded, old, and not easily kept clean	5
New	Fairly clean	3
Poolihari	Better drained than old line, but too crowded	4
Talap	Fairly clean, but old	4
DHANGARI DIVISION:		
Dhangari	Clayey retentive soil, dirty lines	6
Singli	Scattered, lying fairly high on sandy soil	2
Nowgong	Lowlying marshy jungle with bad water supply	10
Runiamutti	Sandy soil, new, and clean ..	None

The figures given are approximate, as the exact population of each line could not be ascertained, but I went into the matter carefully with the native doctor, a most intelligent man, who has been seventeen years on the garden. The table shows that the percentage of cases is in inverse ratio to the cleanliness of the lines.

Nowgong lines, where the proportion is largest, also furnish bad cases of cholera, dysentery and diarrhœa out of all proportion to their size. They are insanitary and are outside the garden and beyond official control.

Talap line seems an exception, as it is fairly clean, but it is very old, and some small jungle lines are bracketed with it.

Runiamutti lines only furnished one case in 1899. Here resided rather more than a hundred brand new coolies, all adults, in brand new lines, on a sandy soil, on the high banks of a stream, with a capital well, good drainage, and clean surroundings. They worked apart from the older coolies on *soil newly reclaimed from the forest*, they were *not acclimatised*, and the nature of the soil was against them, and yet they were absolutely free. Fever they suffered from, but not sore feet.

It is just possible that pani ghao, or some cases of it, has a malarial origin, and that the eruption is a true herpes, similar to the herpes labialis so constantly seen during an attack of "fever." It is much like it in appearance at times. Many of the cases suffer also from fever, and fever and sore feet cases are more numerous during the hot steamy days following rain, and when working in new soil. The point against this theory is, that the tea-house people suffer from fever, but do not get sore feet.

Microscopic examination gave me mainly negative results. I made a large number of preparations of fresh serum, drawn from carefully sterilised vesicles on the second or third day; I stained with gentian violet, mounted in balsam, and examined with a one-twelfth oil immersion lens. In the greater number of cases I found no bacteria at all; in the cases where I did so the contents of the vesicles had become purulent. I examined *young* vesicles to make sure that they had not been broken into and reinfected, as many of the pustular blebs have no doubt been. I could find nothing distinctive. The condition I most often noticed was the presence of small, rather scattered crystals occurring in rods and plates, and resembling those of uric acid.

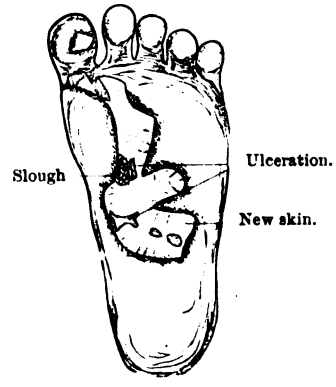


FIG. 10.—VESICULAR TYPE.

Sixth day, showing how extensive an area is often affected. the dead skin has been snipped off with scissors.

Treatment.—This may be divided into *surgical* and *preventive*.

Surgical.—The indications are, to get the case as early as possible; to carefully cleanse the foot by soaking it in warm antiseptic solution; then open the vesicles with sharp pointed scissors, snip the loose skin away, and finally wash the parts with carbolic solution (1 in 40), and treat the resulting ulcer with carbolic oil, phenyle oil, extract of paroli leaf, zinc ointment, &c., according to circumstances. The soaking, washing and dressing operations are repeated once or twice daily, and healing takes place in eight or nine days in favourable cases.

As the number of cases on large factories may run to a hundred or sometimes nearly two hundred daily, large sheds are provided, where the coolies squat in long lines for inspection and dressing. They are taken in batches to have their feet soaked in zinc-lined troughs half full of hot phenyle solution. There are bamboo seats for them, and there they sit for half an hour, when another batch takes their place, whilst they go away to a native dresser who is provided with carbolic solution and the necessary dressings. The simpler cases have the feet covered with phenyle oil, and are left without other covering; the more serious ones are dressed with boro-iodoform, cotton wool, bandages, &c., according to the degree of severity.

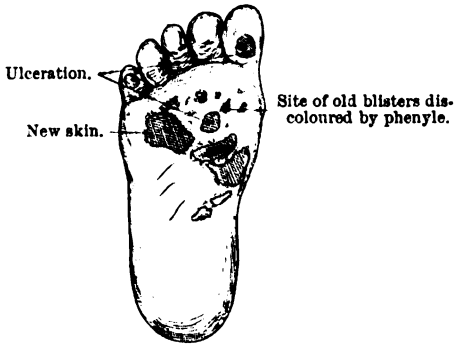


FIG. 11.—VESICULAR TYPE.

One week old. The sores vary in intensity and in different portions of the same sore; in some the epidermis comes away as it does from a blister, in others it is more deeply bitten, and an ulcer results.

On some factories it is the custom to touch each vesicle when opened with pure carbolic acid or a solution of nitrate of silver, with the idea of destroying the irritant which caused the mischief, and of leaving a healthy surface when the resulting superficial slough should come away.

If the case is not got held of early enough, instead of a small vesicle which, when opened, leaves a simple sore like that resulting from a burn of the first degree, we get an ulcer with a slough varying in thickness and extent according to the amount of neglect or the health of the patient. The vesicles may become large blebs filled with purulent fluid, or may burrow more deeply and become abscesses.

The abscesses are opened, drainage tubes inserted if necessary, and treated on general surgical principles. Whenever the feet are much swollen they are soaked in hot phenyle and poulticed, generally with paroli leaf poultices, and the foot is rested all day. Sometimes a patient goes to work before the sores are properly healed, and he comes in with the ulcers broken out afresh and worse off than before. He may also have a crop of new vesicles scattered about in between the old sores, or a new crop may come on the other foot. This new crop, too, may occur in a patient who is attending hospital every day and has not been to work in the garden, so that either we have a longish incubation period and a double inoculation, or else the disease was picked up elsewhere than in the garden soil.

Paroli leaf is to be obtained from a jungle tree somewhat resembling the walnut, and in the form of a poultice made by simmering down the leaves in water is the remedy used by the natives of Assam for sore feet. It is also used in many hospitals, and the older coolies use it themselves in their lines.

Preventive.—It has been suggested that the coolies' feet should be dipped in some antiseptic solution before going to their work in the garden. On a neighbouring garden the manager placed shallow troughs of phenyle solution at the entrance of the lines, and all the coolies had to walk through them, but it was difficult to supervise, and no steps were, I believe, made to record the results.

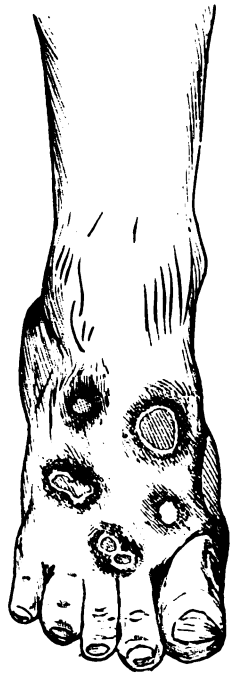


FIG. 12.—VESICULAR TYPE.

Sixteen days attending out-patient department. Large raised indurated ulcers.

I made one experiment in this line. A gang of women, about seventy strong, had their feet painted every morning with a strong solution of phenyle in castor oil, which I thought would be more adhesive than a watery solution and keep out the wet better. The percentage of cases went down a little, and I think the cases which occurred were milder, but the experiment was on too small a scale to deduce much from.

Kurrams are the native wooden clogs worn in some parts of Bengal. They consist of a thick wooden sole with a knob, or a double cord for the toes to catch in, and a high heel at both ends, the whole being generally carved out of a solid block of wood. They keep the feet well off the ground, and if the coolies could only be induced to wear them at their work it would make a vast difference in the number of cases. It would not put an end to the disease, as the hoeing man cannot wear them, he would sink into the ground too much, and new coolies take a season or two to learn their usefulness.

I only know one garden (Sookerating) where the kurram is regularly worn, and the favourable position of that garden on the following table I believe to be due to that cause. In other respects it is like other gardens, and before the custom was established, some six or seven years ago, it was one of the worst gardens in the district for sore feet. I have constantly gone into the hospital there, and on making enquiries amongst the sore feet cases, found that all the men were hoeing men, and all the women new coolies, or for some reason not in the habit of wearing kurrams.

SUMMARY OF SORE FEET CASES DURING MAY, JUNE, JULY, AND AUGUST, 1898-99.

1898.

Name of Garden	Number of Labour Force	Daily average No. of Cases	Daily Percentage	Total No. of Cases
Talap	3,000	73	2·4	
Khobong ..	1,750	27	1·53	2952
Sookerating ..	1,424	14	·98	

1899.

Name of Garden	Number of Labour Force	Daily average No. of Cases	Daily Percentage	Total No. of Cases
Talap	3,000	59	1·96	
Khobong ..	1,750	27	1·5	2914
Sookerating ..	1,519	12	·82	

There were fewer cases in 1899 than in 1898 though the rainfall was heavier, but I put the decrease down to improved surgical cleanliness.

The difficulty lies in inducing the coolies to wear them; they hate new customs, and learn them with reluctance. The manager of Talap made some thousands of clogs one year, and had to burn them in the engine furnace, as the coolies would not wear them.

On *Khobang* they wear them to a certain extent, but the kind supplied consisted only of a piece of board shaped to the foot, not so effective as the kind with double heels.

The number of cases are fewer in proportion than on Talap, but more than on Sookerating where proper kurrams are worn.

A HANDY METHOD OF PREPARING SLIDES AND SLIPS FOR TAKING BLOOD FILMS.

By W. L. Braddon, F.R.C.S.

Negri Sembilan, Malay Peninsula.

The attention of workers is drawn to a simple method of preparing slips and slides for blood examination, which I have been in the habit of using for some time.

The (sterilised) slides and coverslips having been thoroughly cleaned, they are to be dealt with as follows:—

(*a*) *Preparation of slides with coverslips attached.*—The slip is placed on the slide, in such a position that one of its edges coincides exactly with that of the slide, and is firmly pressed. Then, for temporary use, vaseline, for permanent purposes white cement, is smeared over all the edges of the coverslip, except that which corresponds with the edge of the slide, and a small portion of the edge opposite.

(*b*) *Preparation of paired coverslips.*—Two slips, preferably square ones, being accurately superposed and firmly pressed together, the vaseline, or cement, is smeared over all the edges except one which is left free, as before, and a part of the edge opposite.

(*c*) *Mode of use.*—On touching with the free edge of a slide, or paired slip, so made ready the drop to be examined, the blood enters in a thin film in which the corpuscles are spread out with beautiful uniformity, having suffered a minimum of change from exposure to air, and none at all from handling or pressure. When the whole space has been *slowly* filled, the edges and small opening previously unsmeared with vaseline or cement are closed by applying a little of the former with a small camel's-hair brush. Fresh blood so taken will keep longer than after any other process with which I am acquainted. Stains are best added by placing a drop of them on the surface from which blood is drawn, and puncturing through the drop. The latter should be small, and it is well to let the blood run in between the surface slowly.

The method has the advantage that the slides or slips can be used by the most unskilful; and will stand a great deal of knocking about without injuring the specimens.

The paired slips are the more useful, in that one pill box will accommodate material for collecting scores of specimens. The necessity of cleaning new slips from time to time, preserving them in alcohol, and burning them off in a flame when required for use—always more or less of trouble in domestic practice, and out-country work—is also avoided. All that is necessary is to keep the slides ready in small grooved boxes, the slips in pill boxes, in layers with pieces of blotting paper between them.

IV.

GOUNDOU AND AINHUM IN SOUTH CHINA.

By J. Preston Maxwell, M.B., F.R.C.S.

Shangpoo, S. China.

The pathology and exact diagnosis of the above two diseases, are as yet not fully settled and they are supposed to be the peculiar possession of the African. And although a case of ainhum has been reported from Swatow, goundou has up to the present time been supposed to be absent from China. For this reason the two following cases may prove interesting.

The patient in the first case, that of Goundou, is a woman aged 45, mother of several children, none of whom present examples of her disease. She is extremely superstitious, so much so, that neither money nor any other means, spread over a year and a half, have enabled me to obtain a photo, and I have to be content with a sketch drawn after careful observation. The appearance of the woman is typical, so much so that seeing her in the street at once recalled the pictures of the disease. The swellings are symmetrical, more rounded than oval and in the situation shown in the drawing. They are hard, painless and

the skin over them is movable and natural. There is no epiphora or discharge from the nose, but I was not able to make an examination of the interior. As far as I can learn, headache has not been a marked symptom. The disease began when she was a girl, steadily progressed for some years, is now entirely stationary and the woman appears to be in good health.

GOUNDOU, FROM A DRAWING.

The case of AINHUM, which is illustrated by a photograph, is interesting in more respects than one.

The man, a Chinaman, aged 44, came to me eighteen months ago with a condition precisely

AINHUM (?), FROM A PHOTOGRAPH.

similar to ainhum. His left little toe was swollen and nearly surrounded by an ulcerated groove. This ulceration showed some tendency to spread more widely on the plantar aspect of the foot. The disease had begun in the interval between the fourth and fifth toes. The stunted third toe is *not* due to disease, but to an accident occurring some twenty years previous. Under treatment the diseased condition much improved, and the man went home. At this time there was no anæsthesia about the legs and no sign of leprosy. He did not come from a family infected with the disease. About two months back he returned presenting the condition depicted in the photograph. The groove around the little toe was mostly healed, but it had spread in the opposite direction, and the fourth toe had been removed by it; there was also a small ulcer on the dorsum of the foot over the head of the fourth metatarsal, and there was also some ulceration on the plantar surface in this situation. The skin over this surface and around these toes was in a hide-bound condition, but there was still no affection nor anæsthesia of the skin of the legs. But after another month I discovered an anæsthetic slightly scaly patch on the extensor surface of the leg on the affected sides, and obtained from the patch a bacillus having the shape and form of the leprosy bacillus.[1] Since that time fresh patches, erythematous, anæsthetic, and non-sweating, have appeared, and there is no doubt that the man is a leper. In view of the obscure pathology of the disease in question, the above case is important. Some time ago my medical colleague, independently of any suggestion from myself, asked if I did not think that it was a case of ainhum, and *now* there is no doubt that the man is a leper. What the connection between the two diseases may be I am not inclined to dogmatise, but strange to say, text books altogether shun the question of the presence or absence of the leprosy bacillus in the cases which have been already recorded.

July, 1900.

NOTES ON BLACKWATER FEVER AS FOUND ON THE CONGO (MID-CONGO STATE).

By Mr. CHAS. B. BANKS.

MY first personal experience of blackwater fever came to me after about one year's residence in the Congo, and was accompanied by jaundice, the temperature reaching about 105° F. The blackwater lasted in this fever about thirty-six hours, the fever for about three weeks; convalescence was slow. Within the next two and a half years I had three other blackwater fevers. I then returned to England, where I remained over a year. A few months after my return to Congo, I got another blackwater fever; this fever was a pretty bad one and pulled me down considerably, but convalescence was rather quicker than in some of my previous attacks.

During eighteen years of Congo life I have had twelve or thirteen blackwater fevers. All were preceded by pain in back and loins; and almost all by some fever which pulled me down far more than the temperature or length of fever accounted for. Moreover it always left a feeling of malaise and unfitness for work, although the thermometer showed a normal temperature; then within a fortnight fever came on and

[1] All attempts to obtain this from the ulcer had failed.

blackwater showed itself in from ten to forty hours after the commencement of the fever. Sometimes the blackwater would be accompanied by jaundice and continuous vomiting; in other cases there would be no jaundice and very little vomiting. The urine at times would be clear and bright red, at other times very dark red, sometimes so dark that it required to be diluted with water to show the red colour. I have seen fevers on the Congo when the water became almost black, but when diluted with water and the glass held up to the light appeared bright yellow. This, although sometimes mistaken for blackwater fever, is not the same, being caused by enormous quantity of bile in the urine, and of course requires different treatment. The duration of the blackwater is very uncertain, sometimes it may stop for a few hours or even days and then begin again. My last blackwater fever continued for three and a half days; it then stopped for a few hours and began again, and lasted for twelve hours; then stopped for a week (the fever still continuing) and appeared again for about twenty hours, but disappeared for about five hours (water quite clear); it recurred for ten hours, after which it kept away for nine days (fever still continuing) when it came back again and lasted for twenty-four hours; each time after the week's intermission the temperature rose to 105·5° F. This fever lasted for over five weeks and left me prostate.

In the case of other white men whom I have treated, they almost always had a little fever before the blackwater appeared. Some maintained they had had no fever, but only felt a bit out of sorts; but in these cases the temperature had not been tested by thermometer. Sometimes it appeared to come on immediately after a chill.

There is a peculiar odour emanating from blackwater patients as a rule, which enables one, together with the peculiarly anxious and worried expression so often seen in the faces of these cases, to recognise this disease (or stage of disease) even before an examination of the urine. When the blackness of the water is caused by bile this peculiar odour is absent.

I have had considerable experience among patients suffering from this disease—over a hundred—all of whom recovered.

Although what is known as blackwater fever has generally been considered a separate disease, I have treated it as ordinary remittent fever; when jaundice has manifested itself together with blackwater, as bilious remittent; the blackwater I have treated as simple hæmorrhage from the kidneys, arising nearly always from congestion, and to stop this I have used calomel and jalap first, then ext., or tinct., of hamamelis, or hazeline, or the hazeline alternately with the extract. If this did not improve the character of the urine within a few hours, I repeated the calomel and jalap, and always found the urine change for the better after the second or third dose of calomel and jalap. I have given as many as twelve doses of calomel and jalap during one fever, and always with a good result. Some cases I have only given one dose, but as a general rule three to four are needed.

When first I see the patient I always try to get some quinine into the system as quickly as possible; sometimes I give it together with the calomel and jalap, and have found this plan answer well; but after blackwater has manifested itself I only give about 5 to 10 grs. night and morning, but after the blackwater has stopped I increase the dose to 15 grs. of the bisulphate. Where I find it necessary to continue quinine for a long time in large doses, I prefer it dissolved in nitro-hydrochloric acid, as when thus given it does not disturb the digestive organs so much.

I control the temperature with phenacetin or antipyrine, sometimes in conjunction with a little quinine; but I only use these drugs when the temperature keeps persistently above 102·5° F.

The vomiting, which is sometimes so persistent and troublesome in this disease, may often be stopped by 2½ to 5 grs. of Dover's powder, followed in a quarter of an hour or twenty minutes with a dose of calomel and jalap disguised in jam. When I wanted to give quinine and could not get the patient to keep it down, I found the hypodermic injection of the bi-hydrochlorate answer very well, and never saw any trouble arise from its use in this way, unless the needle or syringe used were in an unclean state.

I have always made it a rule when I had a bad case of fever to attend to, to keep a close watch on the temperature, and so, when I could do so without unduly disturbing the patient, took the temperature every hour night and day; one point of difference in the temperature has often decided me as to what treatment to adopt. By doing this I have often been able to recognise the threatened recurrence of blackwater, and by giving medicine at once greatly shortened its duration. When I found quinine had a bad effect upon the kidneys I gave ol. eucalyptus, 10 min. night and morning, and found it acted well on the kidneys, besides acting as an antiperiodic. It is especially beneficial where there is uræmic poisoning threatening.

I am inclined to believe that blackwater is only a complication arising in the course of a remittent fever and should be treated as such. I think it is often the result of sudden congestion of the kidneys, and sometimes kept up and aggravated by such drugs as quinine, antipyrine, phenacetin. My reason for believing this is that I have always found it ceases when steps have been taken to thoroughly relieve the kidneys and hæmostatics given. I have sometimes used ergot ext. with good effect, although the temperature generally rose a little after its use.

I have noticed it is generally persons of a nervous temperament, also those of a gouty or rheumatic diathesis who are most prone to have blackwater fever, and it generally comes on after a time of worry or anxiety.

MOSQUITO-SCREENED HOUSES *VERSUS* QUININE.

By A. H. HANLEY, F.R.C.S.I.
District Medical Officer, Southern Nigeria.

MANSON, Ross and others, have ably demonstrated that anopheles are capable of infecting man with malaria, and that the use of the mosquito net at night minimises the likelihood of infection if it does

not, as on the Roman Campagna, entirely prevent it. A member of the Liverpool Malarial Expedition attributed his immunity from fever during four months' residence in Southern Nigeria, not to the taking of quinine, but to the careful use of the mosquito net at night. Anyone who goes to Southern Nigeria and expects to steer clear of fever by use of the mosquito net at night will, I fear, in many cases be soon disillusioned. The anopheles here attack you both day and night. In my practice out here I have had to do long journeys in a boat at night, and for twenty-six miles have been exposed to the attacks of mosquitoes and other pests; but, thanks to ten grains of quinine taken either when starting or on my arrival, I have for years escaped fever. After fifteen years' experience of Southern Nigeria, I cannot speak too strongly of quinine as a prophylactic, and still advise all comers to take quinine as heretofore till such time as the place in which they reside is free of anopheles. My reason for writing you is that I see a tendency to put the quinine bottle on one side and depend on the mosquito net. The people who do that, either do not believe in quinine as a prophylactic, or fail to see that the mosquito net at night confers but partial protection.

BODIES IN THE URINE IN A CASE OF BLACKWATER FEVER.

By E. G. Hamilton Williams, M.R.C.S., D.P.H. and Mary Hamilton Williams, M.B., B.S.Lond., D.P.H.Cantab.
Special Service Medical Officers to the Ashanti Field Force.

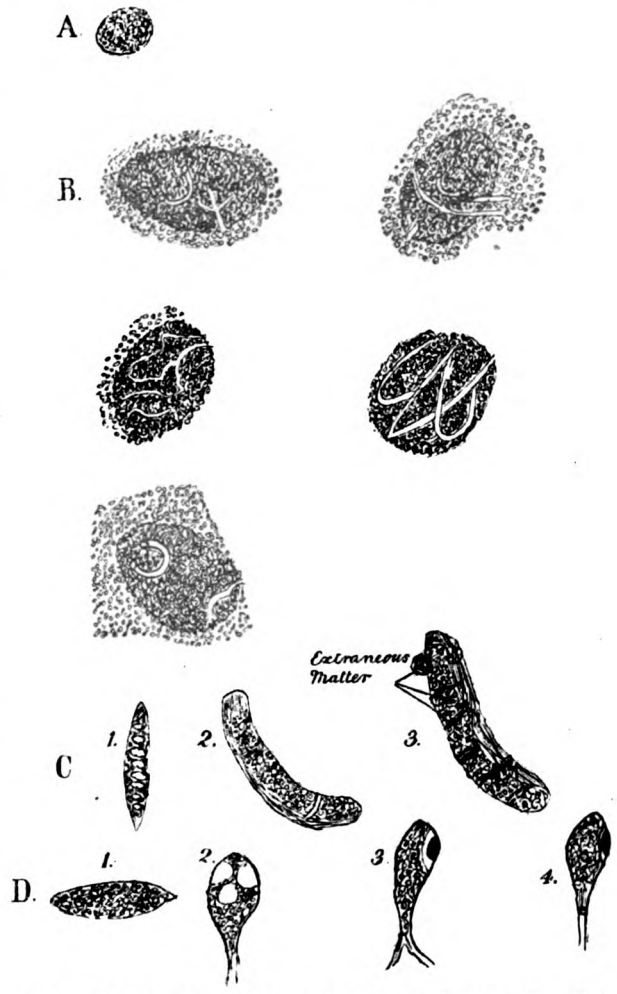
The following case presented the common features of an attack of blackwater fever. An officer, who had had slight fever for several days, vomited repeatedly during the night, and on looking at his urine noticed that it was the typical port wine colour. The colour gradually faded, and in five days the urine appeared normal.

The urine on the first day presented the usual characteristics described in the text-books. On examination of an unstained slide, with Zeiss "D" lens, nothing of note was detected, but as we found afterwards, it is possible, when familiar with the bodies described below, to detect them with this power. They then appear as small pale yellow or colourless bodies, with an ill-defined round or elliptic contour, sometimes containing one or more refractile spots.

The following observations were made either with a $\frac{1}{12}$-in. oil immersion lens and 8 eye-piece, or an $\frac{1}{18}$-in. oil immersion and 4 eye-piece.

The urine was kept in stoppered bottles for about six hours and a drop of the sediment then placed on a slide and allowed to dry in a film, passed quickly through the flame, then stained with methylene blue. Various other stains (fuchsine, gentian violet, aniline blue, Bismarck brown, safranin, &c.) were used, but methylene blue was the most satisfactory.

There were apparently two kinds of bodies [? organisms] not present in ordinary urine: A.—A body of an elliptic or round contour, showing a refractile spot [? vacuole] with a central dark spot; the rest of the body finely granular, or containing fine vacuoles, stained faintly blue, with a distinct margin. B.—Far more numerous bodies, round or elliptic in contour, staining deep blue, with an indistinct margin, surrounded partially or completely by an irregular area of some substance stained less deeply. The substance of the bodies appears to consist of fine granules or vacuoles, but within each body can be seen a more or less complex tube or tubes, these showing as non-stained portions. In some it can be made out that the end, or ends, of this tube (or tubes) communicate with the exterior and in a few appear to be continued for a short distance outside the body. In others, a



A, B, and C are drawn to a scale twice as large as D. For description of diagram see text.

fairly frequent arrangement resembles that in vorticella, viz., horse-shoe shape, with a refractile spot at each end. In a few cases it appears as if the granules within the body were especially distinct near the margin and even become continuous with the granules in the faintly stained area surrounding the body, as if they were being extruded from the body into this area. Either the lightly or darkly stained bodies (A and B) may occur alone, or in groups of three or four. In a few cases they look as if attached to each other.

In the second day's urine the results of the examination were the same as that of the first, but the number of bodies was smaller. The third day's urine showed a very small number of these bodies and also a few bodies like those marked C 1, 2, 3.

The urine of later days showed nothing of note. A few drops of the first day's urine were added to a test tube of water—repeated examination of this showed no new body. Small quantities of the first and second days urine were kept in stoppered bottles. On examination on the eighth day of sediment from these urines, the bodies marked as D 1, 2, 3, 4 were seen, in some parts of the slide grouped together in dense masses. Each is a somewhat pear-shaped body, with a dark spot placed laterally on the broad end of the pear, and partially surrounded by a clear space. In some a refractile spot is seen near the stern part. A few, with larger heads, contained unstained, or faintly stained portions which occasionally looked as if they might be red blood cells, *e.g.* in D 2.

The question arises, are any or all of these bodies organisms connected with the disease? The writers have never seen anything resembling them in any other urines, and would very much like to know if any other observer has described them. In the article on blackwater fever in "Albutt" it is stated that Dr. Manson has discovered certain oval bodies in the urinary sediment, but few if any of these now seen are exactly oval. The large numbers found of A, B and D make it appear to the writers probable that they are not accidental, but it is mostly the similarity in structure of the bodies marked C which make any relationship of these likely. Are the bodies described various stages in the life of an organism, and if so, is it pathognomonic of blackwater fever?

NOTES AND OBSERVATIONS ON DISEASES OF THE TROPICS.

By O. Johnson, M.D.

Lagos, West Africa, forwarded by H. E. Sir Wm. McGregor, G.C.M.G.

Diseases in the tropics have much the same features in common with diseases of the same kind in other parts of the world; but as climatic and other natural causes have a powerful bearing on disease, we may, therefore, expect to find distinctive features of even the same class of disease in various zones and climes. The habits of the people, especially as regards food, dwellings, and other sanitary matters, must also be taken into consideration; hence we find that some diseases are peculiar to certain countries, as are the people inhabiting them.

In speaking of tropical diseases, the following remarks apply chiefly to Western Equatorial Africa. In the tropics, the seasons are generally described as *two*; viz., the wet and the dry; the most sickly period is the interval between the two seasons, especially during the change from wet to dry.

Malaria.—Malarial fever is by far the most common disease of the tropics; the large amount of study that has been spent upon it leaves nothing to be said on the subject here; but I may observe that natives of West Africa are conversant with many kinds of herbs and drugs acting as preventives or curative agents, of these, I may mention:—

(1) *Lime juice.*—During the fever season, fresh lime juice is used as a preventive; about a wine-glassful taken the first thing in the morning will prevent an attack of fever for that day at least. Whenever an attack is expected, and the premonitory symptoms of an onset threaten, a dose of fresh lime juice will always be attended with a favourable result.

(2) *Powdered cobwebs.*—On the Gold Coast, the natives use the cobwebs of a certain species of spider as a cure for malarial fever; they wash, dry and powder it; and when taken in the same measured doses as quinine, it is attended with a similar result. I have not used it myself, but I know Europeans who have tried it, and spoken well of it.

Malaria may be the cause of much unhealthiness and want of energy, but cannot be said to be a fatal disease to natives of West Africa.

Enlarged spleen is well-known to be a concomitant of malaria; but one fact to be noted in connection with this is, that chronic enlargement of the spleen is invariably found among mulattoes. It may be stated as a fact that there is no mulatto in whom the spleen is not more than twice the ordinary size. I know of some in whom the spleen overlaps the liver, and yet they seem to keep in fairly good health.

Blackwater fever is, I suppose, classed among tropical diseases, although as a matter of fact, it does not generally attack natives of the tropics, but European sojourning in the tropics. This dreadful malady is accountable for a large proportion of European deaths in the tropics. Blackwater fever is, in my opinion, wrongly attributed to malaria; probably because it is often complicated with malarial fever; but any attempt to treat it purely as such, must lead to lamentable results.

That natives do not suffer from it, is a proof that it is not due to malaria, as all alike suffer from this, although in different degrees. We may note also that mulattoes as a rule do not suffer from it either; in this, however, we should make a distinction; mulattoes who are brought up as Europeans, do suffer from it, but I know of no case where they succumb to it as Europeans do; whereas those who are brought up as natives enjoy the same immunity as their native brethren.

In the early days of the West African Colonies, blackwater fever was scarcely ever heard of; its notorious prevalence is of later date; this fact has led some to attribute it to over dose of quinine, as it was almost unknown in the pre-quinine days; but if it is due to quinine, this would not limit its attack to one class of people, but would affect equally all who indulge in large doses of quinine. Too large doses of quinine probaby aggravates the malady, or increases the discomfort and irritability attending the fever, and therefore cannot be trusted as a remedy.

In my opinion, the cause of this malady is to be looked for in the habits of the people who suffer from it; by this I mean, the European mode of living, chiefly *their diet.*

Animal food forms a large proportion of the diet of Europeans, and this distinguishes it broadly from the diet of Africans. Animal food is very stimulating,

Fig. 1.

Fig. 2.

Fig. 2.

Fig. 3.

Fig. 3.

To illustrate Dr. OSLER'S Case of Multiple Gangrene in Malarial Fever.

Bale and Danielsson, Ltd., London.

and, to live well in the tropics, it should almost disappear from the general diet, especially during the hot season. I advance this view with diffidence, but from experience, I have been convinced that the cause of this blackwater fever is to be sought for in that direction.

When we consider that solids derived from animal food are largely eliminated by the kidneys, we can imagine how, in the hot weather, in a country where the skin acts so freely—a large proportion of the liquid being eliminated by the skin—the solids, if ingested in a large quantity, are left to block up the delicate urinary tubules, and consequently suppression of urine, rupture of the urinary tubules and hæmorrhage, which are the common features of this disease, follow with fatal consequences. The consuming thirst which generally accompanies this disease is due not only to the thirst of fever, but to the loss of blood and want of liquid to flush the kidneys; hence a very sharp purge, unlimited amount of bland drinks, and sinapisms on the loins often bring speedy relief to the sufferers.

So far as I am aware, this theory as to the causation of blackwater fever has not been advanced by any one, but I should like attention to be given to this view as to its correctness.

The excessive consumption of alcohol as predisposing to blackwater fever has also been advanced by some. This may or may not be the fact, but my opinion on this subject is that the evil done by strong drinks when taken frequently as a beverage, is that it destroys the resisting power to any disease, especially fevers, in which its deleterious effects are most marked.

Pneumonia and *consumption* are not peculiar to the tropics, but they are responsible for a large amount of mortality.

Pneumonia, I may mention, is peculiarly fatal to Kroo boys. They are known to succumb to it rather more rapidly than other people do.

The modern treatment of consumption known as the open-air method, has not, to my knowledge, been tried on any large scale, but I may mention that I know of two cases at least of perfect cure by that method, one of which was under my own care.

Yaws we always regard as a disease due to filth. It is almost peculiar to the Ijebus and Aworis, tribes inhabiting the lagoon littoral near Lagos. They regard its influence as benign, asserting that an attack of yaws will expel all other diseases from the subject, so that one who has suffered from yaws is considered a perfectly healthy individual. The common mode of expressing a good wish to one another amongst the Ijebus is, "May the yaws visit your family." This is regarded as praying for that perfect health which follows the supposed benign influence of yaws. This disease does not yield to the same treatment as for syphilis which in other respects it resembles so much, but to scrupulous cleanliness and wholesome diet. It always and invariably leaves behind it a chronic eczema plantaris.

The *sleeping sickness* is, I believe, peculiar to West Africa. It is always fatal. There is still ample room left for the more satisfactory study of this peculiar disease.

The *ring-toe*, or, as the Yorubas call it "ayùn" (*i.e.*, a file), is also peculiar to West Africa. This disease does not cause death, but disfigurement. It always attacks the small toe, appearing as if a strong cord is tied tightly round the toe. It is very sensitive and painful to the touch; nothing seems to have any influence to check its progress, the constriction will go on and on, until the toe drops off or is severed. The cause of this disease is also unknown. I know of a case in which it is hereditary in three generations.

Smallpox differs from the other diseases in that it is an object of worship. It is regarded as a visit of the god, and therefore more efforts are spent in propitiating the deity than in finding the means of cure. The beloved of the gods will always get over it, and the guilty sinner must succumb.

It is a custom to supply palm wine as drinks in large quantities to the sufferer. This being a good diuretic, the effect of it, in a case like this, in which the action of the skin is in abeyance must be beneficial. But there are those who take a more rational view of this disease, and what is called native vaccination, a powerful preventive and ameliorative derived from vegetable products, is known and practised by a few as a secret remedy, and seems very effective.

Dysentery, as we have learnt, accounts for a larger proportion of deaths than any other disease. In practice we find some cases yielding easily to sedatives followed by an astringent, and others require antiseptics (*e.g.*, hydrar. perchl.) for their cure.

As typhoid fever is practically unknown it always appears to me that cases which in northern climes will break out as enteric, in the tropics will assume the form of dysentery.

Elephantiasis[1] is probably not so common in West Africa as in other countries in the tropics. Two cases have come under my care which I think can never be surpassed for size. The patients could not bear their own weight, and consequently could not walk. One was at Sherbro in Sierra Leone and the other at Lagos. The former, who was only two days with me in the hospital, was simply brought down from up country as a witness in a criminal case, and was merely passing on to the capital for the Assizes. I was only able to relieve him of two bucketfuls of water (hydrocele fluid), so that he was able to walk out of the hospital by himself.

The other case was in the Lagos hospital, when I had charge of it in 1895. I operated on the patient and he left the establishment quite well.

Tetanus is by far the most dreaded disease in the tropics. It is very fatal, especially in the traumatic form. It has recently been asserted that all forms are traumatic, but, of course, it is not in all cases that the wounds are obvious. One thing, however, is clear, that it is more prevalent (in both forms) at certains seasons of the year, notably from the middle of July to the middle of October.

Guinea Worm is one of the most widely prevalent diseases of the west coast. A large amount of quackery has been devoted to it, but the only thing I know that has any really modifying effect on it is assafœtida. It certainly moderates the pain and promotes

[1] E. Græcorum.

the extraction of the parasite. Free poulticing and lancing of the abscess are the best means of speedily getting rid of the parasite.

Considering the notorious unhealthiness of the West African Colonies it is a matter of surprise that hitherto no distinct Health Department has been formed in any of the colonial governments, and no direct means of research into the causes of disease. This defect has happily been remedied in our colony of Lagos. From this we may hope for much, and we trust we may prove *able seconds* to institutions established for the same purpose in England. The discoveries made of the propagation of malarial fever by means of mosquitos have opened a vast field of enquiries as to the causation of other diseases by similar agencies. The mangrove flies and sand flies are known to be intolerable pests in the lowlands of tropical countries, and it is not unlikely that we may find them also to be agents in disseminating diseases.

In conclusion, I beg to suggest that, as there are at least half-a-dozen medical men always resident at Lagos, a Medical Society be formed, with the Chief Medical Officer as President, and a certain amount of co-operation be permitted and encouraged in researches into the causes and cure of tropical diseases.

A CASE OF MULTIPLE GANGRENE IN MALARIAL FEVER.

By William Osler, M.D.
Professor of Medicine, Johns Hopkins University.

There are three groups of cases of multiple gangrene:—

(1) *Raynaud's Disease.*—There have been previous well-marked vascular disturbances in the extremities (syncope, asphyxia or hyperæmia), the gangrene is very often symmetrical, is usually slight in extent and limited to the fingers or toes, more rarely to the ear-tips or nose.

(2) *Multiple Spontaneous Gangrene of Limbs.*—In young or middle-aged persons, without any obvious cause, massive gangrene of one, two or three extremities occurs. Many illustrations of this are recorded in the literature.

(3) *Multiple Spontaneous Gangrene in Association with the Acute Infections.*—In measles, typhoid fever, typhus fever, scarlet fever, diphtheria and malaria, local gangrene may occur. There are multiple patches, not symmetrical, and the skin and subjacent tissues are more frequently affected than the extremities. While of course the phenomena of Raynaud's disease may occur as a sequence of any of the specific fevers, a large proportion of all the cases of local gangrene occurring during or after one of the fevers have nothing whatever to do with this affection.

The relationship between malarial fever and Raynaud's disease is believed to be very close. Many references are given to cases (a majority from French sources) by Barlow in his article in Albutt's System, and more fully by Monro in his excellent monograph on the disease (Glasgow, James Maclehose and Son, 1899). Altogether, in the cases he has collected, there were only 8·3 per cent. with malarious antecedents. I have looked over the notes of cases of Raynaud's disease which I have seen in Baltimore, nine in number, and I do not find malaria to be related as an etiological factor in any one of them, nor, so far as I know, in our very large series of cases of malaria during the past ten years has there been a single instance of Raynaud's disease.

The following case is a very remarkable illustration of multiple gangrene occurring in a case of æstivo-autumnal malaria. Similar cases have been reported in the literature, and are referred to by Monro in his monograph (page 96), but they seem to be exceedingly rare.

Clinical Summary.—*Malaria when 6 years old—typhoid fever twice—last attack four months before onset of present illness—illness in the middle of October, supposed to be influenza, but more probably malaria—on November 2 onset of spots of gangrene in various parts — rapid extension — condition on admission as shown in the figures—complexion muddy—spleen enlarged — blood showed very many æstivo-autumnal organisms—temperature slightly elevated at first—subsequently no fever—rapid recovery.*

P. W. B., aged 23, bar-tender, admitted to Ward E, Thursday, November 29, 1899, complaining of sores on various parts of his body.

Family History.—Mother died of consumption. No history of rheumatism or of any special disorders of the skin.

Personal History. — As a child he had measles, mumps and whooping cough. When 6 years old he had malaria. Five years ago he had a very severe attack of typhoid fever, after which he had an abscess in the abdominal wall, which opened spontaneously and discharged for two mouths, leaving a large scar. He had at the same time many boils. Last year he went south with the Fifth Regiment, and in August he had a second attack of typhoid fever, and was ill for two months. He has had gonorrhœa twice; has never had lues. He has used tobacco freely; whiskey and beer in moderation.

Present Illness.—The patient has been living in Baltimore this autumn, and has been very well until the middle of October, when he was ill in bed for nearly two weeks with pains in the back and general weakness; no fever, no chills, no herpes. The doctor called it influenza. The patient got up and was about for a few days, when, on November 2, just twenty-seven days ago, he noticed blebs about half an inch in diameter on both hands, which were slightly swollen. The next day a mottled area appeared on the instep of the left foot. It had a bruised appearance. A similar one appeared on the buttocks and on the dorsum of the right foot. Other spots came in the situation to be subsequently mentioned.

The hands and feet became very much swollen. The blebs broke and discharged a dark fluid; the skin around the affected areas was very red. There was no itching. He had some pain at night. Ten days ago he had slight chilly feelings. There had been no redness, nor swelling, nor blueness of the fingers or toes, and there had been no numbness or tingling. The urine had been clear. Dr. Futcher made the following note on the day after his admission.

"The patient is a large-framed well-nourished

man; complexion rather sallow. The skin of the whole body is pigmented, markedly so about nipple and umbilicus, to slight extent about genitalia; no increase in either axilla. The lips and mucous membranes are of fairly good colour; no pigmentation of mucous membranes. Over dorsum of left hand, just behind knuckles, there are four whitish scars, the result of healing vesicles. Over the ring, middle and little fingers there is a brownish yellow discolouration of the skin which is gradually peeling off where the blebs are healing. On palmar surface of same fingers the skin is raised in large blebs. The skin has a brownish yellow colour; and over the ring finger is quite gangrenous, and there is involvement of the subcutaneous tissue. The thumb and index finger are not involved.

"*Right Hand.*—The dorsum of hand is unaffected. On the dorsal surface of first and second inter-phalangeal joints of index, middle and ring-fingers the skin is thickened, brownish in colour, no vesicles. Over the hypothenar eminences on palm is a large area, measuring 5 by 6 cm., in which the skin is loosened from the subjacent tissue, markedly discoloured, and at one point a serous fluid is exuding. The palmar surface of all four fingers shows a gangrenous condition of the skin with vesiculation and oozing of fluid, most extensive on ring finger, where the process invades the palm of the hand.

"*Right Foot.*—Over dorsum of foot, below ankle, is an area, 5 by 3 cm., in which the skin is gangrenous and exceedingly black; slough still adherent to adjacent tissue; surrounding skin slightly pigmented. Over the heel there is an area of brown, discoloured, thickened skin, measuring 5 by 6 cm.; this area is sensitive to the touch.

"*Left Foot.*—Below external malleolus is an area, 5 by 3 cm., of gangrenous and sloughing black skin.

"*Left Buttock.*—Just over the spine at the junction of the dorsal and lumbar regions there is a patch of dry gangrenous skin 1½ by 2cm. Over left gluteal region there is an irregular gangrenous patch, quite dry, measuring 4½ by 2 cm., slightly sensitive to pressure.

"*Occiput.*—Over the lower part of occiput, on each side, there are two areas in which the scalp has a gangrenous appearance, slight oozing of fluid causing matting of hair."

Though the history did not suggest malaria, as in the routine examination of the abdomen the spleen was found to be considerably enlarged, the blood was at once examined, and very large numbers of æstivo-autumnal organisms were found. The crescents were in unusually large numbers. Cultures taken from the blood proved negative. There was no leucocytosis, and the differential count was practically normal. The eosinophiles were only 2 per cent. The patient was at once given quinine in full doses, and he began to improve rapidly. The larger sloughs were treated with linseed poultices made with bichloride solution. Crescents and ovoids persisted in the blood for some time, though by December 15 they were rapidly disappearing. On December 14 the gangrenous patches on both hands had healed. On the feet the sloughs had separated, leaving deep ulcers, the sheaths of the tendons being exposed. The urine examinations were negative throughout. The patient had a slight rise of temperature (100°) at first; subsequently none at all. The figures from photographs, by Dr. Brownell, illustrate the condition on admission.

[We are indebted to the courtesy of the Editor of the *Johns Hopkins Hospital Bulletin* for this paper, and the loan of the blocks illustrating the paper.—Ed. *Jl. T. M.*]

SURGERY IN THE TROPICS.

Lecture at the London School of Tropical Medicine.

By James Cantlie, M.B., F.R.C.S.
Surgeon Seamen's Hospital Society.

A medical practitioner in the tropics is expected to be an adept in all branches. He not only is expected to be able to recognise all kinds and phases of disease, but is to treat them in the light of the most recent research. Whether it be an ovarian tumour, a cerebral abscess, a glaucoma, or an enterectomy, he is expected to produce results equal to, or in view of local prejudice better than, the first surgeons of Europe. With this responsibility thrown upon him, and I have only mentioned the surgical aspect of the case as being most apparent to the lay eye, what is the result? I do not claim for such men that they, as a general rule, can rank with those whose genius has established our principles of thought and action in surgery and medicine, but I do claim that they represent a class of practitioner who has almost died out in Europe—a man whom specialism has driven out and buried, and who never will or can appear again. No doubt the present state of affairs is occasionally to the advantage of the patient and to the advancement of knowledge, but with it has gone the individuality and the responsibility attaching to the medical man himself.

The tropical practitioner is a general practitioner in the best and truest sense of the word, and the effect is that we have in the colonies and in many up-country parts, medical men with an experience in clinical work and in operative surgery such as is not obtainable in any hospital in this country. Called upon to treat on his own responsibility every or any ailment in the category of disease, he is unwittingly made self-reliant, courageous, and deft in the practice of his profession in a way unknown to those dwelling near larger centres and seats of learning.

One of the first duties of the practitioner when he proceeds to a warm climate is to provide himself with instruments of the very best quality. This may be the case also at home, but it is doubly so when one is far away from surgical instrument makers, or even from those who can repair simple defects in mechanism. A faulty lithotrite, which fails when the critical moment comes, will risk the patient's life and bring discredit upon the practitioner's skill and upon European surgery generally in the eyes of the hypercritical, doubting and suspicious native. The surgeon is held responsible for his instrument as well as for his skill, and a bad instrument may turn the native prejudice against him, and cause a loss in prestige and money which may mean ruin. As an example of the kind I will

relate a circumstance which occurred to myself. Whilst performing lithotrity on a Chinaman of influence and position a lithotrite I was using gave way and I had to lay it aside. I had provided myself with a second, and thought myself fortunate in having had the forethought to thus meet eventualities. What was my chagrin when the second instrument also became unworkable owing to the catch on the screw also getting out of order. Two instruments with the names of well-known makers stamped upon them had failed me and I was left in a dilemma. I proposed lithotomy, but the friends would not hear of it, and the patient had to be allowed to recover from the anæsthetic. Luckily the accident occurred in Hong Kong, where several large hospitals equipped with all surgical requirements are near by, and I was able to obtain a third instrument wherewith to complete the operation. Had I been operating in one of the out-lying stations my patient would have no doubt refused further treatment, and a severe blow to surgery and to my practice would have ensued. There is a belief that tradesmen in this country when they want to get rid of their goods, especially those that are old shop keepers, send then to the colonies. That this is the case with some of the surgical instrument makers I can hardly believe, but the circumstances I have related behoves the surgeon to look well after his instruments.

All rubber and caoutchouc instruments and appliances have but a short period of perfection in tropical climates, especially in those that are hot and damp. The climate very soon spoils soft and hard rubber material, and I can find no written suggestion as to how in any way to prevent or even delay the destruction. So serious is this deterioration that it at times may affect the nature of the operation to be performed. I will relate one or two. I had just performed two consecutive lateral lithotomies, and was proceeding to a third, when at the suggestion of a few of my colleagues standing by I was persuaded to perform the suprapubic operation. Just as I was about to open the bladder the rectal bag burst. A second bag was being inflated when it also burst, and a temporised rectal distension had to be employed. This was one of the few cases of lithotomy I ever lost, and I cannot help attaching a good deal if not the entire failure to a faulty bag. Old tubing kept as carefully as may be is a constant worry and anxiety in surgical practice in warm climates. A piece of tubing of the kind caused me considerable trouble in a case of liver abscess I was draining. The tubing seemed all right when first introduced, but after ten days in the wound it broke off during manipulation. Rubber of course in every country perishes, but in tropical countries its period of usefulness is but short lived. Many advise to keep the rubber in water, or again, to moisten it with oil. The rubber part of the lithotrity bottle is in the same category, and it is with an anxious look the surgeon regards the deep fissures in the rubber part of the bottle whilst attempting evacuation of the fragments, in case of their giving way. We look to our instrument makers to advise us in these matters, and to inform the profession what are the best steps whereby to preserve rubber material in the tropics.

The surgeon in the tropics, in all but the large centres, has not infrequently to conduct major operations even single-handed. He may manage to impress some more than usually courageous layman into his service, who may give a temporary help; but it not infrequently happens that he has to keep his eye fixed occasionally on his assistant to see that the colour is not deserting his lips and face, or his attention may be only drawn to his condition when he sees him lying on the floor in a faint. Instead of a European friend the surgeon may have a native trained to assist him, and he in time comes to rely upon him with more or less confidence. But at times—and these are usually the most important occasions—the mechanical help which an intelligent native brings fails, or even his intelligence causes him to meddle with instruments he but imperfectly understands. I hope it has never occurred to many of you, but I will give an example of such a calamity. An aspirator is an instrument which excites the curiosity of the native, and he delights to handle it. But the principles of pneumatics are not included in his education, and he cannot gather that air is a fluid that can be increased in density or exhausted by extraction. Hence it may happen, indeed it has happened, that the surgeon when he inserts the needle of the aspirator finds that the air enters the chest with, it may be, fatal consequences instead of the fluid being drawn off. Dr. Manson related to me an eventuality of this nature. When preparing to use the aspirator, he instructed his native assistant to exhaust the air from the bottle. After vigorous pumping the taps of the instrument were turned on, and just as he was about to insert the needle the cork of the bottle was blown out with a loud report. No doubt the timely explosion saved the patient's life.

The keeping and preserving instruments of a metallic nature from the effects of the climate is a constant care in a tropical climate. Endeavour as one will, and be as careful as one may, rust will creep in to an extent that those who are not acquainted with what a hot, damp climate is, can scarcely believe. It not unfrequently happens that new instruments are corroded when they arrive fresh from the surgical instrument makers at home. However tightly fitting the box lid may be, and however carefully wrapped in cotton wool, there is no excluding the penetrating effects of damp during an ocean voyage. When enquiry is made at the instrument makers they show you a box lid that fits with microscope closeness; they tell one how carefully the box itself has been dried before being sent, and they even take the precaution to expose the instruments themselves and the cotton wool around them to dry heat, so that no particle of moisture can be present in the box when it leaves their hands. I may say at once that all this is to little purpose unless the box itself is enclosed in a soldered tin cover. Damp, especially sea damp, in the tropics will penetrate any crevice, however microscopic. If a resident in India wishes his cigars to preserve their flavour, they must be sent in soldered cases, and so must surgical instruments. A surgeon practising in the tropics is justified in sending back any case of instruments arriving from home in a case that is not satis-

factorily soldered; and by the time the instruments have reached the makers, when they have been exposed to yet another spell of sea damp, the maker will no doubt, when he opens his case, be impressed with the fact that perhaps the surgeon, after all, had right on his side when he remonstrated against their not being sent in a hermetically sealed case.

Every instrument that can be made of steel, or metal of some description, instead of wood or other material, should be so made. What the direct effects of the climate may not accomplish the tropical cockroach will, for to him neither wood nor caoutchouc come amiss. A soft catheter is to him a delicacy, and even the silver linings of plated instruments are food for this voracious animal. Corrosion and rusting may no doubt be delayed by smearing the surface of instruments with oil or lard, carbolic oil or an ointment of some sort; but any hollow instrument will corrode inside as well as outside, and it is impossible in some to prevent it. The hypodermic syringe is an instrument which is at all times and in all countries a troublesome one to keep in order, but I am convinced that our tropical brethren find this applies with fourfold intensity in warm climates. Needles will corrode and become stopped at intervals which recur with intermittent regularity. If a little pains were taken, however, with the material of which it is made there need not be that constant difficulty. Hypodermic needles made of steel should be utterly excluded from tropical use. In spite of care they will become blocked, and even if the wire is passed along it before the practitioner can finish his rounds so that he may well vaseline the wire, it may be too late, and the wire is found to be immovable in the needle. Needles of gold are much less likely to corrode, but gold is too soft a material for the purpose, and it is apt to break off at the insertion of the metal into the holder. Platinum or nickel are the best material and the only material which should be used in hypodermic needles sent to the tropics, and the surgeon should return promptly any instrument in which steel forms a part.

The drying or expansion of the piston buffer is another source of trouble and annoyance, and the material of which it is made is an important point. Nor should the barrel in any part consist of vulcanite. Given a hypodermic syringe of glass and vulcanite and steel needles, and you have an instrument in every particular as it should not be. I here show you an instrument which in every particular is suited to tropical requirements. It is made of metal in every part, piston, buffer and barrel. The needles are of platinum or nickel. It is strong, cannot deteriorate by climate, and cannot get out of order. It may be expensive, but it will repay the money laid out upon it in a sense that no other instrument will. Surgical instrument makers must be brought to see that they must supply only the best material for tropical use, it will pay them in the long run. This may be true in a general sense at home, but it is so in a very special sense in practice in out of the way parts of the earth, and for an instrument to fail a man so placed may mean loss of life, as diseases are much more acute in most tropical countries than in those of temperate climates, and the fact of a hypodermic needle failing when it is wanted may be attended with serious, or it may be fatal, consequences. The surgeon also who is providing himself with an armamentarium before proceeding to practise in the tropics should be impressed with the absolute necessity of having the best there can be got, and not be content to take the first instrument that is foisted upon him, its chief recommendation being frequently the beauty of the velvet garb in which it is set. No velvet trappings must defile the instrument case intended for the tropics. Metal alone must be the setting in which surgical instruments are placed.

Treatment of Snake Bite.—In the *International Medical Magazine* for September, 1900, Professor McFarland sums up the treatment of snake bite as follows:—(1) Stop immediately the circulation in the bitten member or part of body, so as to prevent absorption of the poison. (2) Incise and enlarge the fang wound freely and suck forcibly to extract the poison—the suction may be accomplished with a surgeon's cupping glass or with the mouth, the poison being harmless when swallowed. (3) Inject hypodermically 3 to 6 drops of a fresh 10 per cent. aqueous solution of calcium chloride into about a dozen different areas about the wound. Gold chloride is just as effective, but is too expensive. Potassium permanganate is of little value. (4) Give strychnine hypodermically to stimulate the respiratory centre. Whisky should not be given at all, or only in very small doses, because an excess of alcohol still further depresses the heart already depressed by the venom. (5) *Immediately* inject 10 to 20 cc. (2½ to 5 fl. dr.) of antivenomous serum and repeat these injections frequently. The author advises people living or going into regions where there is danger of snake bites to carry a bottle of antivenomous serum with them.

Carbolic Acid in the Treatment of Pneumonic Plague.—Dr. J. Bell, of Hong Kong, sends a communication to the *Lancet* on the subject of treating pneumonic plague by carbolic acid. Pure carbolic acid was administered every three hours in 12-grain doses, and strychnine and digitalis hypodermically every four hours. The treatment was continued for four days, during which period 280 grains of carbolic acid were given. The case reported by Dr. Bell is the first case of pneumonic plague known to have recovered.

Climate.—Travellers who have not seen "Climate," in *Quarterly Journal of Health and Travel*, edited by C. F. Harford-Battersby, M.D., should lose no time in procuring a copy. In the Journal will be found useful and practical information on many points it is essential for them to know.

The Journal of Hygiene.—We welcome this new Quarterly Journal to be published in January, 1901, by the Cambridge University Press, and edited by George H. F. Nuttall, M.D., Lecturer on Bacteriology and Preventive Medicine in the University of Cambridge. In Dr. Nuttall's able hands a Journal of Hygiene is sure to be a publication of high merit, and must command the attention of all to whom Hygiene, in its widest sense, is of interest.

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THE

Journal of Tropical Medicine

DECEMBER, 1900.

THE SECTION OF TROPICAL DISEASES AT THE BRITISH MEDICAL ASSOCIATION.

ALL interested in Tropical Diseases will be pleased to know that the section devoted to the reading of papers, and to the discussions, on diseases of the tropics is to be again held at the forthcoming meeting at Cheltenham. This will make the third year in which the section has met, and judging by the experience of the 1899 and 1900 meetings, a great deal of good to tropical medicine will be the result. In no section of the Association is the work entered into with greater enthusiasm than in that devoted to Tropical Diseases. There is so much that is new, and so much to be done in this department of medicine, that there is no likelihood of the subjects for discussion proving "stale or unprofitable" for many a year to come. We have as yet but touched the fringe of the commencement of the subject. Even malarial studies are but in their infancy, and when we consider the vast fields open for exploitation, it is not the dread of exhausting the special work of such a subject, but the appalling nature of its magnitude, which hampers us. The subject of dysentery and bowel affections may be said to have been scarcely broached, and certainly no broad laws even have as yet been formulated to serve as a guide or scientific basis for study. We hope soon to see this remedied. Sir William Macgregor, in his address at the London School of Tropical Medicine, did well to insist upon the necessity of a closer investigation of these subjects. Malarial commissions we have had; a commission of investigation into the causes of enteric is at present at work in South Africa; beri-beri is being particularly enquired into in the Malay Peninsula, and we hope soon to hear of a commission entrusted with powers of enquiry into dysentery and allied ailments. Dysentery is but a name for a group of ailments; the term has no scientific precision; its limits, cause and relations are practically unknown, and its treatment is for the most part merely empiric.

To yet another subject would we direct attention. Surgery in the tropics is, so far as our literature goes, a neglected subject. Since Sir Joseph Fayrer's classical works on surgery in India were written, little has been done in the way of publication except by Colonel Kenneth Macleod. We would welcome further additions in this field of work. We are accustomed again and again to hear our colonial brethren speak in disparaging terms of the amount of surgical work done in home hospitals as compared with what is accomplished in large hospitals in, say, India and elsewhere. Yet do we get but meagre accounts of this large field of work; and with the exception of the article "Surgery in the Tropics," in volume ii. of Gould and Warren's "International Text Book of Surgery," no standard work has devoted special attention to the subject. We hope practitioners in the tropics will

take up this subject, and from the wealth of their knowledge send contributions on surgical subjects to the Section of Tropical Diseases at the forthcoming meeting of the British Medical Association in July, 1901. We can assure them that their papers will be welcomed and duly appreciated.

Translations.

ON THE RELATION OF THE MOSQUITOES TO MALARIA PARASITES IN CAMAROON.

By Dr. Hans Ziemann, Staff Surgeon (Victoria).
(*Translated from the German by P. Falcke.*)

Report I.

As soon as I arrived in Camaroon, at the commencement of March, 1899, my incessant efforts were directed towards confirming the etiological signification of mosquitoes to malaria. It was known that Camaroon, one of the most dangerous malarial regions, was but little visited by mosquitoes, at least as regards the coastal localities.

Nevertheless, I succeeded gradually in isolating thirteen different kinds of mosquitoes, belonging to either the *Culex* or the *Anopheles* species. The latter, at that time, was only represented on the Joss Plain by an anopheles related to the *Anopheles claviger*. This discovery of the presence of the anopheles on the west coast of Africa was made by me by the end of March, 1899.

I succeeded, besides, in finding out points as to the classification and conditions of life of most kinds, which should be of great consequence in the hygienic measures to be adopted.

As instances of a few of these points, larvæ or pupæ were never found in quickly-flowing water, nor in water which at flood was invaded by sea water, though otherwise, hygienically, these creeks have a bad name. As in India, the larvæ are only found in small standing pools. Larvæ and pupæ of the *Culex* were found in water containing 1·1 per cent. of common salt. A larger quantity of salt seems to check the development of the mosquito. In small ponds covered with scum no further development of the larvæ took place. Thin layers of petroleum quickly retarded further development of the larvæ and even caused them to die off rapidly. The pupæ were always more capable of resistance than the larvæ. Notwithstanding the careful dissection of many hundreds of mosquitoes from the Joss Plain and up the river, I never succeeded in finding forms in the salivary glands and stomachs of newly caught culices or anopheles, such as were discovered by Ross and R. Koch in mosquitoes which had sucked the blood of birds containing proteosoma; nor did I find the so-called crescent and spheroid forms of the human malarial parasites, such as the Italians, Grassi, Bignami and Bastianelli confirmed in mosquitoes which had fed upon the same.

Only the spheroid form can develop further in the stomach of certain mosquitoes. I was never able, in accordance with the above-named authors, to discover a further development of the ordinary ring-shaped parasites of the febris tropica in the stomach of the culex or the anopheles. I soon found reason for the above negative results, firstly, in the fact that the proteosomal infection of the birds which were used for mosquito experiments was here exceedingly rare. Of a total of ninety-six birds, I found this interesting infection in but two, and both birds soon died. Secondly, the transformation of the malarial parasites of this country into the crescent spheroid forms, which alone come into the question for experiments with mosquitoes, is exceedingly exceptional.

I often came near to doubting the correctness of the mosquito theory. I thought with longing and secret envy of the material at the disposal of the Italian investigators, whose patients often had masses of crescents in their blood. Out of more than 1,000 examinations of blood I could only detect on twelve occasions isolated crescents.[1]

On the other hand, embryos of filariæ of the genus *Filaria perstans* were found three times in the intestine of the mosquito.

The above-mentioned difficulties which stood in the way of my investigations on mosquitoes were not rendered less by a very onerous practice. For a long time I was the only doctor in Camaroon. Shortly before the end of my term of office in Camaroon, I found on March 27, 1900, in the stomach of a medium-sized grey culex—the definition of which I reserve—a large number of cocci of the same form and in varying sizes, such as Ross and R. Koch had already discovered in mosquitoes infected with proteosoma. I succeeded, besides, on March 29, in finding in two newly caught anopheles, similar to the anopheles claviger above mentioned, two forms which I considered the earliest adolescent form of malarial cocci.

These discoveries gave me the impetus to strenuously continue the experiments with mosquitoes during the short time at my disposal before leaving Victoria.

After much searching in the houses of white people and in the huts of the indigenous Bakweris, I at last found two infected species of anopheles in the dwelling of a workman on a cocoa plantation. The one corresponded with the kind found on the Joss Plain, the other was one-third smaller, but was otherwise similar to the first. Thirty per cent. of the anopheles there found were infected with younger or older malaria cocci, such as have been lately described by the Italians. One black kind of culex with white rings on the legs, which was likewise found there, was never infected. The three white inmates of the house frequently suffered with febris tropica. In two of them I could recognise crescents, but only periodically and in very small numbers. Experimental infection with culices bred from the larvæ were always negative. On the other hand, I succeeded in infecting anopheles bred by myself by allowing them to suck

[1] Perhaps it is worth mentioning that the season from May to October, 1899, was a relatively healthy time in Camaroon.

blood containing crescent forms. Anopheles which had imbibed no crescents, that is to say, that only sucked healthy blood, never exhibited malarial cocci. Unfortunately a number of the infected anopheles always died, so that therefore my material was very small.

However, the important fact is confirmed that in Camaroon also the parasite of febris tropica may change in the stomach of the anopheles to a pigmented coccus-like form, in the interior of which the so-called sporozoitis develop. The latter finally reach the salivary glands, from whence, by means of the proboscis, they may again be inoculated into another person.

By a lucky chance, a little time after, I had the opportunity of examining blood which contained the parasites of ordinary tertian fever, such as is met with in Germany. The blood examined contained also the so-called spheroid forms of the tertian parasite. I infected an anopheles (kindred to the anopheles claviger) with this blood, and after three days I found that the tertian parasites were transformed (though in limited numbers) into malarial cocci.

From the small quantity of material at my disposal I am not in a position to decide if the malarial cocci of ordinary tertian fever and tropical fever show any difference at this early stage. At all events I have hitherto been able to confirm the action of anopheles in all types of malaria.

The investigations as to the signification of the bloodthirsty sandflies, which in this country are exceedingly troublesome, have hitherto been negative.

As a result of my conclusions a larger scope is afforded for the hygienic measures in Camaroon, and my proposals relating thereto will be embodied in my longer report.

Further investigations will elucidate the point as to whether the bite of the mosquito represents the sole manner of the transmission of malaria, as also whether man is the only vertebrate animal that harbours malarial parasites.

A calm and careful consideration of this question seems to me to be the more necessary on account of the feverish haste with which malarial examinations are often conducted.

I may mention that I succeeded in finding a new blood parasite which greatly resembled the parasites of febris tropica in a small long-tailed monkey, but I could not discover a further development of this monkey parasite.

Five years previously, thus long before the Italian Deonisi,[1] I had confirmed the existence of quite similar parasites in bats in Camaroon, and the same parasites have shortly been found in bats again. But in these also no further development could be discovered on examination of the interior organs of the bats.

[Report II. will appear in a future issue.]

[1] *Centralblatt für Bacteriologie*, 1896.

MAURITIUS.

Annual Report on the Museum for the Years 1898 and 1899.

The report, furnished by the superintendent of the museum in Mauritius, Monsieur A. Daruty de Grandpré, shows that a great amount of useful work has been accomplished during the past two years. The director complains that the limited space at his disposal renders an effective display of the several collections impossible; and those destroyers of tropical museums, white ants, seem to be causing great annoyance.

Sugar Cane Parasites.

In the interests of both the colony and of science generally, the museum seems to be doing good work. The insects injurious to agriculture, more especially those which attack the sugar cane plantations, have been particularly studied. One of the most destructive of these is "a kind of scale insect (*Coccidæ*)," and the museum staff have been engaged in devising means of getting rid of this and other pests. It is found that hydrocyanic gas is a potent antisecticide. To obtain the same end, the *Vedalia cardinalis* (lady birds) have been introduced from Cape Colony, and set free in order to provide a new auxiliary for the destruction of parasites. This is in accordance with the steps taken in California, and it is to be hoped the results will prove equally successful.

Malaria and Mosquitoes.

The investigation of the malarial-mosquito theory has not escaped attention, and the superintendent reports upon the work done as follows:—

"The recent discoveries of Major Ross in India, and those of Bignami and Grassi in Italy, on the relation of mosquitoes with malaria have particularly attracted our attention, and being fully alive to the importance of such a problem, we have undertaken a series of searches on the Biology, Anatomy and Histology of the *Culicidæ*.

"We are now completing our studies in the part which those insects play in the two different human pathological affections (Malaria and Filariasis).

"In our researches made in 1899 we have discovered in Mauritius eight different species of *Culicidæ*, five species of *Culex* and three of *Anopheles*.

"Amongst the *Culex* three specimens only are very numerous and attack mankind. One species only attacks human beings only during the night, and the two others during the day, and this generally outside dwellings. The three species of *Anopheles* very rarely attack human beings during the day; they rather do so during the night.

"One of the smallest of those species is not, or rather very seldom, to be found in elevated places, and has the same area of dispersion as the malaria in Mauritius; this one has proved to be *Anopheles costalis* (*Lœno*), found also at Freetown (W. Africa) by the *Malaria Expedition*. The other species—the biggest—are only to be found in elevated places and are very seldom met with on the sea-shore where malaria prevails.

"The smallest species of *Anopheles* only begin to

make their appearance in number in the months of November and December; and it is probably this species which, further minute searches, will prove to be the direct agent of transmission of malaria.

"Amongst the *Culex*, the species (*Culex anxifer*) which attacks mankind at night is that which transmits the *Filaria* to human beings."

British Medical Association.

A DISCUSSION ON ANKYLOSTOMIASIS.

(*Continued from p.* 101.)

IV.—Oswald Baker, M.D., Lieutenant-Colonel I.M.S. (Ret.), Physician to the Seamen's Hospital, Albert Dock, London.

The contribution to the consideration of this subject which I wish to make relates entirely to a discussion of the prevalence of ankylostomiasis in Burmah. My attention was first drawn to this question by the perusal of Dr., now Sir William, Kynsey's report on anæmia in Ceylon, issued by the Ceylon Government in 1887, of which I was fortunate enough to obtain a copy shortly after its publication. It appeared to me on reading this report that the anæmia so common in Burmah might also, as in Ceylon, be the result of ankylostomiasis. Although the ankylostoma duodenale was not at that time known to exist in Burmah, it seemed highly probable that on investigation this parasite would be found to be very prevalent throughout the province, for both the habits of the people and the climatic features of the country were in the highest degree favourable to its existence and dissemination.

In returning to Burmah, therefore, I lost no time in making an inquiry into the matter; nor had I long to wait, for within a few days of resuming charge of my duties a Hindoo lad was admitted into the Civil Hospital suffering from anæmia in a profound degree, for the production of which there was no obvious explanation. The condition of this patient was practically identical with that of a person who had bled to the verge of death, and the symptoms he presented were precisely those and those only which result from great loss of blood from whatever cause arising. Ankylostoma ova in abundance were at once found in his stools, and on the day following this discovery two of the worms, both dead, were seen in his evacuations.

While the existence of the ankylostoma in Burmah was thus proven, the fact that this blood-sucking parasite was extremely prevalent throughout the province was soon afterwards established beyond doubt. In the Moulmien gaol, of which I had medical charge, so prevalent was the ankylostoma among the prisoners that I seldom failed to detect its ova in cases not only of simple anæmia, but also of anæmia associated with specific diseases. As, however, an investigation limited to convicts already inhabiting the gaol was open to the objection that the prisoners in question might have acquired the parasite subsequent to their admission, it was resolved to examine the stools of a large number of prisoners at the time of admission. Now, the gaol at Moulmien is what is known as a central gaol, and as such receives within its walls criminals sent not only from the town but also many who are brought in from different parts of the province. Obviously, if large numbers of newly admitted prisoners were found infected with ankylostomata, my belief that the parasite was extensively disseminated would receive substantial confirmation. I determined, therefore, with the aid of a trained assistant, to microscopically examine the stools of 100 prisoners admitted consecutively into the gaol as soon as possible after their reception.

Before the observations were fully completed, however, I was transferred from Moulmien to an appointment elsewhere, but the transfer did not take effect until 89 of the proposed examinations had been made. The results obtained may, I think, be accepted as representing the degree of prevalence of helminthiasis among the lower classes of the adult population of British Burmah.

Of the 89 prisoners, 17 were natives of India and 3 were Chinamen. Taking the natives of Burmah alone, of whom there were 69, it was found that no less than 38, or 55 per cent., were infected with ankylostomata; that 33, or 49 per cent., had whip worms; and 30, or 43 per cent., had round worms; while the stools of 2 contained the ova of the tapeworm. Only 10 out of the 69 were without some species of entozoon. So that whilst 55 per cent. harboured ankylostomata, as many as 86 per cent. were the subjects of helminthiasis of some form or another. It may, I think, therefore, from these observations be safely concluded that the ankylostoma duodenale is present in the intestines of at least half of the poorer classes of the inhabitants of Burmah.

That a high prevalence of ankylostomiasis is both directly and indirectly responsible for much mortality there can be no question, but the extent of this mortality unfortunately cannot be ascertained. Numerous deaths take place annually, especially among the lower orders, from "unknown causes;" and in hospitals and gaols the mortality from anæmia and general debility of uncertain origin is often high. Much of this loss of life, set down to unknown causes, to anæmia and to general debility, is, I think, unquestionably due to ankylostomiasis.

I drew attention to this mortality in a short paper on Ankylostomiasis in Burmah, which was published in the *Indian Medical Gazette* some twelve years ago. I further pointed out that an infection with ankylostomata, unless adequate to the production of pronounced anæmia, would probably altogether escape recognition. I also explained that the reason ankylostomata were not always found *post mortem* in fatal cases of ankylostomiasis was due to the circumstance that these nematoids, subsisting on human blood, often abandoned their impoverished and anæmic victims before death, owing to want of sufficient or suitable nourishment. Subsequent observers, amongst whom I would mention Giles, have expressed very similar views.

That ankylostomiasis should obtain so high a degree of prevalence in Burmah can, when the habits and customs of its population are considered, hardly cause surprise. There is practically no night-soil sanitation in the outlying villages of Burmah, and most of the inhabitants defæcate on to the ground through holes in the floors of their dwellings. Left on the surface of the soil, exposed to wind and rain, a portion of this excrement becomes spread broadcast over the land, whilst some of it, in the form of mud, is carried into the houses of the people, and is deposited on their floors.

Now the majority of the Burmese do not use chairs and tables at their meals, but squat on the floors and eat their food with their fingers out of plates and bowls set down on the floor in front of them. When travelling they often dispense altogether with feeding utensils, and sometimes substitute the leaves of certain trees.

Under such circumstances the general prevalence of intestinal entozoa is, I would affirm, inevitable.

Kynsey has maintained that water is undoubtedly the chief source of infection in ankylostomiasis, and has stated that the prevention of this disease consists essentially in the use of pure water. I do not think that such is the case. On the contrary, I believe the infection is conveyed through the medium of contaminated food, and that water has but little to do with the matter. In support of this opinion I would point out that among the 89 prisoners already referred to there were, as stated, 17 natives of India and 3 Chinese. Now these men, both Indians and Chinese, are far cleaner feeders than the Burmese, although the source of their water supply is the same.

Of these twenty aliens only two were found infected with ankylostomata, being 10 per cent., as against 55 per cent. of the Burmese. Moreover, Giles has shown that water retards and finally arrests the development of ankylostoma embryos. There is, Giles states, "no condition more hostile to them than when immersed in water."

I would take this opportunity of pointing out that I believe the spread of many other entozoa is effected in precisely the same manner, that is through the medium of food and not of water. In the town of Moulmien, which in regard to sanitation is probably the most neglected town in the whole of Burma, infection with ascarides is almost universal. About every third person applying for treatment in the out-door department of the Civil Hospital is an applicant for worm medicine. The water supply is mainly derived from uncovered shallow wells, of which many are without parapets, and which in seasons of drought often run dry. I have taken water from one of the dirtiest of these wells when nearly dry, have filtered it and subjected the residue to microscopical examination without finding therein a single ovum or embryo of any entozoon.

It would seem therefore, from a consideration of these dates, that no marked diminution in the prevalence of and mortality from ankylostomiasis in Burma can be looked for until some system of sanitation has been generally adopted which will obviate the broadcast distribution of infective night soil. And to this question of night soil conservancy the attention of the authorities concerned should, I think, be given.

V.—Patrick Manson, C.M.G., F.R.S., LL.D., M.D., Physician to the Seamen's Hospital Society, Greenwich.

Dr. Patrick Manson said it is somewhat singular that rhabdonema intestinale is so seldom found by Giles in Assam, considering that it is so frequently associated elsewhere with the ankylostoma, and that the climate and other physical conditions closely resemble those of Cochin China, where the parasite is extremely common, and where, indeed, it was first discovered by Normand. I have frequently met with it in the stools of Indians. I have twice encountered the ankylostoma in dangerous profusion in Englishmen from abroad. One case came from the West Indies. I had him in hospital for many months, and he was ultimately discharged as hopelessly ill with pernicious anæmia. Fortunately after his discharge as an incurable he consulted a practitioner in the country, who found ova recognised to be those of the ankylostoma. Under treatment with thymol the patient got quite well. A second case came from Singapore. Ankylostomiasis had been diagnosed, but specific treatment was postponed until it could be undertaken under more favourable climatic conditions. When I saw the patient he was profoundly anæmic, as in advanced Bright's disease, with vertigo, tinnitus, palpitation, and all the symptoms of advanced anæmia. Every slide of his fæces showed numerous ankylostoma ova. Under thymol he rapidly recovered, and left for the East again quite well. I am astonished at Dr. Fearnside's statements as to the therapeutic impotence of thymol. In my experience it rarely fails, provided it be given in adequate and rapidly repeated doses, say 30 gr. every hour, for four times. The diagnosis of malaria from ankylostomiasis by hæmocytometic methods, though interesting and valuable from a pathological point of view, is impracticable under ordinary circumstances. Dr. Rogers's statements as to the presence of a leucocytosis during the acute stage of malarial infection are not in accordance with recent observation. Perhaps I misunderstood his meaning; but the presence of a leucocytosis is generally held nowadays to exclude a diagnosis of malaria. I am disappointed that none of the papers have alluded to Powell's interesting observation that befel nut-chewing is possibly a protective habit in the natives of Assam, Burmah, and the Eastern Peninsula, acquired instinctively in consequence of its prophylactic virtues against the ankylostoma. I agree with Colonel Baker's remark that the parasite is generally acquired in food and dirt rather than in water, and that improved methods of night soil conservancy are much to be desired on this account in the East. I believe the Chinese method of dealing with night soil to be a good one on sanitary grounds. The soil is stored in cemented tanks, where it rots and ferments for months before it is used as a fertiliser. During this period of storage it is probable that the ova and embryos of the intestinal entozoa it may contain are killed.

VI.—Major Ronald Ross, I.M.S. (Ret.), Liverpool School of Tropical Medicine.

Major Ronald Ross remarked, regarding the life-history of the parasite, that he had carefully studied Major Giles's experiments on a rhabditiform stage, had partly followed them and considered that they were sound. He thought that Sonsino's criticism of these experiments was unsound. Sonsino said that Giles had mistaken rhabdomena for ankylostoma; Major Ross supported Giles in stating that the former are uncommon in Assam—at least in Nowgong, and thought that such a mistake is too obvious a one to be easily made by a competent observer. He held that the well-known fact of the disease being so commonly an earth-worker's disease supported Giles's discovery. He had long maintained the opinion regarding its mode of infection just given by Colonel Baker, and thought that there is little to be said in favour of infection by the route of drinking water. Regarding the clinical effects of the parasite, he scouted the idea that it is always harmless. It is impossible to fix the exact number of parasites required to produce pathological reaction. Rogers's estimate of 500 is probably much above the mark in many cases. Obviously, numbers required to produce reaction must vary inversely as the strength of the host. Fifty ankylostoma might destroy a patient already debilitated by other disease or starvation. The speaker considered that the parasites may also produce a toxic effect, and referred to Daniel's discovery of yellow pigment in the organs of cases of ankylostomiasis—examples of which he had seen. He concurred with the observers in thinking that the worms may leave the patient in the last stages of the disease. He entered a plea for the much more general use of microscopes for the detection of the ova in the localities where the worms are prevalent, and cited the instance of a hospital assistant in charge of a dispensary full of cases of ankylostomiasis who had not even heard of the disease, and who was treating the cases for malarial cachexia.

VII.—James Cantlie, M.B., F.R.C.S., Surgeon Seamen's Hospital, Royal Albert Docks, London.

Mr. James Cantlie stated that, stimulated by the researches of Dr. Walker in Borneo in connection with the presence of the ankylostoma in beri-beri, he had carried out a careful investigation on the subject in Hong Kong in 1893, but failed to find either the ova or the fully-grown parasite amongst Chinese coolies suffering from beri-beri.

TROPICAL LIVER ABSCESS.

THE MANAGEMENT OF LUNG LESIONS CONSEQUENT ON LIVER ABSCESS.

By Colonel Kenneth MacLeod, LL.D., M.D.

Professor of Clinical and Military Medicine, Army Medical School, Netley.

Of all the routes by which spontaneous evacuation of abscesses of the liver takes place the pulmonary route is the most common. Randus's statistics show that among 159 cases in which liver abscesses discharged spontaneously, 59 or 10·5 per cent., opened into the and through the right lung, and 31, or 5·5 per cent., into the right pleural cavity; or, taken together, in 16 per cent. of these cases the pointing of the abscess took place upwards, through the diaphragm and into the thoracic cavity. The selection of the route in

preference to others depends mainly upon the situation of the abscess, the principle of progress in the direction of least resistance coming into play. Whether the abscess bursts into the pleural cavity or irrupts into or erodes the lung, so as to establish a secondary abscess in that organ, must depend upon the extent and strength of protective pleuritis to which the progress of the pointing gives rise.

When the discharge into the pleural cavity is primary, the event is indicated by characteristic physical signs, and can be very readily and safely certified by exploration. In such cases there is but one indication and rule of treatment, which is to make a free opening into the pleural cavity, removing, if necessary, a bit of rib, and to establish efficient drainage. The opening must be made and the drainage conducted under strict antiseptic precautions. It happens occasionally that the discharge of a hepatic abscess into the right pleural cavity is a secondary event caused by the bursting into it of a lung abscess of hepatic origin, or by a wound of the pleural fold in exploring or cutting into a liver abscess. In these cases the cavity and its contained material are apt to be septic. The same rule of practice governs them; but the drainage must be made as efficient and free as possible, and for this purpose resection of a portion of rib is advisable. A case in which this practice was attended with success under very critical circumstances is related in the *Indian Medical Gazette* for January, 1892 (p. 19).

When the abscess finds its way into the substance of the lung, symptoms indicative of diaphragmatic pleurisy and consolidation of the lower lobe of the right lung accompany, or are superimposed upon, the hepatic symptoms. Should the latter be well marked, exploration through the liver and direct drainage by incision, and the insertion of a tube on ascertaining the presence and situation of an abscess, constitute the proper treatment; but frequently the expectoration of a large quantity of pus is the first positive indication of the existence of an abscess, the preceding hepatic symptoms being masked or obscure, and the exploration delayed. This event may occur after some severe exertion, or in consequence of a bad fit of coughing. The material first coughed up may be very bloody, like an emulsion of brick dust; but sooner or later its character undergoes a change, and it presents the appearance of the flaky, chocolate-coloured stuff known as "hepatic pus." The discharge is generally sudden and profuse. The event is usually preceded by a ticklish cough and basal lung symptoms, and is succeeded by an improvement in the hepatic symptoms, a fall of temperature, and a feeling of considerable relief. In favourable cases the expectoration gradually diminishes in quantity, becomes scanty and finally ceases; the physical signs indicate amendment of the lung lesion, and the patient makes a complete recovery. In other cases the expectoration increases rather than diminishes; or, having decreased, it suddenly increases in quantity as if the abscess cavity had refilled or a new abscess had burst and sought escape through the old channel. This occurrence may take place after an interval, long or short, of apparent recovery, and the second bursting may be preceded by a renewal of hepatic symptoms, hectic fever, &c. The material in such cases is apt to be browner than before, and there is often a considerable admixture of blood. Examination of the sputum gives evidence of disorganisation of lung, and physical examination discloses a cavity, which may be very large and partly filled with air so as to give an area of tympanic resonance. Fever of a hectic or septic type sets in or becomes intensified, and great emaciation and weakness ensue. The exhaustion is increased by the incessant cough and profuse expectoration, depriving the patient of rest and sleep. Death eventually occurs from blood poisoning, asthenia, and choking. A similar sequence and result often arise from neglected empyema eroding and excavating the lung.

(*To be continued.*)

Reviews.

CONTRIBUTION TO THE QUESTION OF MALARIA. Vol. i., MALARIA AND MOSQUITOES. Berlin: O. Salle. 1900.

Carl Schwalbe, well known by his works on malaria, is opposed to the theory of malaria being caused by mosquitoes, and contends that malaria is originated by poisonous gases contained in the atmosphere; moreover, he asserts that the well-known form, which are looked upon as malaria parasites, are not living organisms at all, but forms of degeneration, which are also to be seen in other illnesses and which may be experimentally originated in the blood.

In contradistinction to the above opinion, the observations of this author are interesting and valuable in respect to drinking water and the mosquito theory. New and convincing evidence is brought forward to prove that malaria cannot be conveyed by drinking water, and the author quotes from the current literature and also draws his conclusions from his own vast experience in every part of the world.

To disprove the mosquito-malaria theory, Schwalbe brings forward a series of observations of which the most interesting is the presence and absence of malaria in contiguous localities, localities which are equally visited by mosquitoes. The value of these observations is, however, minimised by the lack of information as to whether the same or different species of the mosquito are present in the various localities spoken of.

Schwalbe's book is very interesting, but he must not labour under the mistake that he has destroyed the value of the mosquito-malaria theory by his sole observations. Be it sufficient for him that he has raised some points not as yet quite covered by the malaria-mosquito theory.

Medical News.

ANNUAL REPORT OF THE HEALTH DEPARTMENT OF SHANGHAI, CHINA, FOR 1899.—The report of the Health Department of Shanghai, by Dr. Arthur Stanley, is quite a model in its way. The report is well got up, well printed, carefully written, and illustrated by several photographs. It is satisfactory to note that plague has not gained a footing in Shanghai. Sanitation seems well cared for in Shanghai. The Public Health Department has under its charge the Bacteriological Laboratory, the Analytical Laboratory, the Vaccine Station and the Pasteur treatment for bites of rabid animals. In each department useful work has been accomplished. From the Vaccine Station 5,000 tubes were issued during 1899 and the efficiency is guaranteed. Typhoid fever seems the greatest scourge in Shanghai; out of a foreign population of 5,510 persons 108 contracted typhoid. It is satisfactory to note that of this number only six died.

New Drugs, &c.

SULPHAQUA, A USEFUL BATH FOR RESIDENTS IN THE TROPICS.—Sulphur baths have attained a permanent and world-wide reputation in the treatment of many cutaneous ailments. In Europe the populations of several fair-sized cities are maintained by visitors who go to them for purposes of the "cure." The natural springs have a virtue which does not belong to a mere mixture of sulphur with water, and the difficulty has been to procure an imitation of the natural solution. The Seltzogene Patent Charges Company of St. Helens, Lancashire, seems to have solved the problem, and by the ingenious production of a nascent sulphur for immediate use have largely done away with the necessity for spending time and money to reach and reside at Homburg, Harrogate, or other fashionable (sulphur) watering places.

To residents in the tropics *Sulphaqua* will be peculiarly welcome. Skin diseases in warm climates, especially those of a vegetable parasitic nature, are prevalent to a degree unknown in Europe. That prevalent pest, "Dhobhie itch," constantly threatens; pemphigus contagiosus, pityriasis versicolor, &c., are diseases of ever-recurring frequency; and not to mention the animal parasites and skin diseases to which prickly heat renders the skin liable, the nascent sulphur bath, ensured by the use of Sulphaqua, is a parasiticide which will be welcome indeed.

Another fact, which the manufacturers of Sulphaqua seem to have been ignorant of, is that nascent sulphur is a protection against mosquito bites. The Anopheles, the malarial-bearing mosquitoes, cannot live in a sulphur-impregnated atmosphere, and the use of nascent sulphur in the bath, especially when it is taken after the heat of the day is over, will help to ward off these disease-bearing insects during the hours of the early evening, when they are most persistent in their attacks.

The fact that the two ingredients necessary to produce the bath are deliquescent, will necessitate their being so wrapped up as to prevent the possibility of the dampness of warm climates proving deleterious to their strength. We have no doubt the manufacturers of Sulphaqua will see that this proviso is attended to.

Letters, Communications, &c., have been received from:—

B.—Dr. A. A. Boucaud (Trinidad).

C.—Dr. J. Cross (Leyton); Dr. M. R. Charlton (Montreal).

F.—Dr. L. Fabian (Trinidad).

H.—Dr. A. H. Hanley (Nigeria).

M.—Mr. J. H. Murray (Fiji); Dr. Patrick Manson, C.M.G. (London); Sir Wm. Macgregor (London).

P.—Prof. W. W. Podwissozzi (Odessa).

S.—Dr. M. Smith (New Malden); Dr. H. T. Strachan (Brockley).

T.—Dr. R. M. Townsend (Rhodesia); Dr. S. W. Thompstone (Old Calabar).

Z.—Dr. H. Ziemann (Wilhelmshaven).

EXCHANGES.

Annali di Medicina Navale.
Archiv. für Schiffs u. Tropen Hygiene.
Archives de Medicine Navale.
Archives Russes de Pathologie, de Medec., Clinique et de Bacteriologie.
Australasian Medical Gazette.
Boletin de Medicina Naval.
Boston Medical and Surgical Journal.
Bristol Medico-Chirurgical Journal.
British and Colonial Druggist.
British Journal of Dermatology.
British Medical Journal.
Climate.
Clinical Journal.
Clinical Review.
Giornale Medico del R. Exercito.
Hongkong Telegraph.
Il Policlinico.
Indian Engineering.
Indian Medical Gazette.
Indian Medical Record.
Janus.
Journal of Balneology and Climatology.
Journal of Laryngology and Otology.
La Grèce Médicale.
Lancet.
Liverpool Medico-Chirurgical Journal.
Medical Brief.
Medical Missionary Journal.
Medical Record.
Merck's Archives.
New York Medical Journal.
Pacific Medical Journal.
Polyclinic.
Public Health.
Revista Medica de S. Paulo.
South African Medical Journal.
The Hospital.
The Medical and Surgical Review of Reviews.
The Northumberland and Durham Medical Journal.
Treatment.

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2.—Manuscripts sent in cannot be returned.

3.—As our contributors are for the most part resident abroad, proofs will not be submitted to those dwelling outside the United Kingdom, unless specially desired and arranged for.

4.—To ensure accuracy in printing it is specially requested that all communications should be written clearly.

5.—Authors desiring reprints of their communications to the JOURNAL OF TROPICAL MEDICINE should communicate with the Editors.

6.—Correspondents should look for replies under the heading "Answers to Correspondents."